DENTAL SURGERY NOTES

BY

ERNEST B. DOWSETT

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ERNEST B. DOWSETT, L.R.C.P., M.R.C.S., L.D.S.

DEMONSTRATOR OF DENTISTRY AND TUTOR OF DENTAL REVISION CLASSES TO GUY'S HOSPITAL

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PREFACE

I HAVE no hesitation whatever in adding to our list of Dental Literature what is intended to be a condensation of Dental Surgery, but I feel that the production of such a book on such a subject necessitates a slight explanation.

Although there may be many who object theoretically to the principle of presenting any subject in condensed book form, nevertheless practically, as a teacher of Revision Classes, and as a tutor for the Final L.D.S. Examination for the last nine years, I have long felt the want of such a book as I have endeavoured to compile. And the many students that have passed through my hands have so frequently asked me to produce in book form the notes I have been in the habit of giving them, that it is principally with the object of assisting them in their much stocked curriculum, that I venture to present this note-book. I have always believed in strict classification and tabulation as an aid to learning, and therefore I have endeavoured to make them prominent.

I have not attempted to detail the ordinary routine

of Conservative Dentistry, as that does not lend itself to any condensation.

Apart from the intended utility to the Dental Student, I hope also that the book may be of some service to the practitioner at times for easy revision or reference.

ERNEST B. DOWSETT.

January 1909.

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DENTAL SURGERY NOTES

CHAPTER I

DEVELOPMENT OF THE JAWS

Look up in latest B.D.J's.

Maxilla

From 7 *centres*—5 Primary.
2 Secondary.

Primary :—

1. Facial surface.
2. Posterior part of alveolar process.
3. Orbital plate.

} 7th week.

4. Palatal process.
5. Intermaxillary bone.

} Few days later.

Secondary :—

1. Nasal process.
2. Malar process.

All join up by 10*th week*, when bone consists of 2 portions :—

1. Larger part made up of 6 centres.
2. Premaxilla.

These separated by suture, which closes about *time of birth*.

Mandible

Developed principally from *Membrane*, but partly from *Cartilage.*

Meckel's Cartilage.

A transitory cartilage formed in the first Branchial Arch. It disappears as ossification progresses, but small piece persists as the *malleus*, except which it has entirely gone by end of *7th month.*

Centres :—

1. "*Dentary*"—on outer surface of Meckel's cartilage, just below future mental foramen.—For body of the bone—5th week.
2. "*Splenial*"—in membrane on inner surface.—For inner walls of sockets.
3. "*Mento-Meckelian*"—for chin.
4. For *Condyle.*
5. For *Coronoid process.*
6. For *Angle.*

Condition of Jaws at Birth :—

1. Halves of mandible separated by fibro-cartilage, allowing free movement.
2. Alveolar depressions show wide open spaces, from which teeth drop out in dry specimens.
3. At posterior walls of sockets of centrals are shallow depressions for pulps of successors.
4. Condyle on level with alveolar margin.
5. Coronoid process makes angle of 135° with body of jaw.
6. Inferior dental nerve and artery seen at bottom of sockets of lower molars.
7. Large shallow depression for 1st and 2nd temporary molars in common.

8. Shallow depression extending backwards for 1st permanent molar germ.
9. Angle little below level of inferior margin of jaw.
10. Antrum is mere depression on outer wall of nasal fossa.
11. Only thin plate of bone between orbit and mouth.

Teeth at Birth :—

1. $\frac{3}{4}$ crown of central temporary incisors.
2. $\frac{1}{2}$ crown of lateral temporary incisors.
3. Tip of temporary canine.
4. Grinding surface of 1st temporary molar.
5. Cusps in form of a ring of 2nd temporary molar.
6. Anterior cusp of 1st permanent molar commencing.

3 Months after Birth.

Teeth do not fall out in dry specimens, as orifices of crypts are closing over.

8 Months after Birth :—

1. Eruption of central incisors by absorption of anterior wall of process.
2. Bony union of halves of mandible.
3. Condyle rising.
4. Antrum occupies inner $\frac{2}{3}$ of floor of orbit, and thus considerable space between orbit and mouth.

After 8 Months of Age.

Age of jaw ascertained by position of teeth.

Dates of eruption approximately :—

Centrals . .	7 months.
Laterals . .	9 „
Canines . .	18 „
1st molar . .	12 „
2nd molar .	24 „

Lower teeth generally erupt little before upper ones, except perhaps the laterals.

Method of Growth of the Mandible

1. In Length.

Alveolar process and jaw made longer by absorption in front of coronoid process, and deposition of bone on posterior aspect of vertical ramus, from the condyle to angle of the jaw, actual observation of which can be seen.

2. In Height.

Jaw deepens vertically by alveolar process growing upwards with the teeth :—

a. With temporary teeth (some of this absorbed).

b. With permanent teeth.

Also before age of 7 years, jaw deepens slightly by deposition of bone on base of jaw.

Above can be proved by comparing sagittal sections of jaws of varying ages. Taking the inferior dental canal as the fixed point, it will be seen that up to 7 years the bone is deposited *above* and *below* the level of the canal, and after that age only *above* the canal, which remains after 7 years at an unvarying distance from the base of the jaw.

3. In Thickness.

Posterior plate of mandible remains unchanged, growth going on by bone thickening from within outwards. This is known by the following measurements, &c. :—

a. If a jaw of a 9 months' fœtus be placed over an adult jaw, it will be seen that the *inside arches* of both are the same. (This is a rough-and-ready method of showing that the jaw grew in thickness by deposition of bone on the *outside*.)

b. Line joining septum between 1st and 2nd temporary molars on either side remains constant at all ages.

c. Line joining middle point of the above line with superior genial tubercles remains constant also.

d. If an adult jaw be shaved down from the outside in the region of the mental foramen, until it is the thickness of a jaw at birth, it will be found, by measuring with a pair of "dividers," that the two mental foramina are the same distance apart and looking in the same direction (forwards) as in the jaw at birth. As the bone is deposited upon the outside of the young jaw, the mental foramina and canal change their direction, passing outwards, upwards, and backwards.

All the above measurements tend to prove that bone was deposited on the outside of jaw.

CHAPTER II

ERUPTION OF THE TEETH

A. Temporary Dentition.
B. Permanent Dentition.

A. ERUPTION OF THE TEMPORARY TEETH

When a tooth is about to be erupted, active absorption of its crypt begins, particularly on its anterior surface, bone behind remaining for part of crypt for successional tooth. As soon as the crown has passed through the free orifice so formed, bone is deposited and loosely surrounds the neck of the tooth, keeping pace with the elongation of root.

Front teeth are erupted first and then back ones; for if all were erupted at same time, teeth could only be closed at the back of mouth. Hence "*proper mutual antagonism.*"

About 6th year temporary teeth all slightly separated from each other and pushed forward, *possibly* by increased size of crypts for permanent teeth.

Theories of Eruption :—

1. *Lengthening of Root*—evidence against :—
   a. Tooth with fully formed root may not be erupted.
   b. Canine travels greater distance than length of its roots.
2. *Growth of Bone at bottom of Socket*—none demonstrable.

3. *Constant's Theory*—increased vascularity of membrane at growing base; thus engorged vessels force up tooth by *blood pressure*.
4. *Coleman's Theory*—"Bone Currents"—*i.e.* presence of a tooth causing bone to grow in its vicinity.
5. *Contraction of Fibres of Dental Ligament*—can only be an adjunct to some other force.
6. *General Growth of Bone*—carrying teeth to the surface.
7. *Delabarre's Theory*—analogous to a Gubernaculum; the fibrous remains of the neck of the enamel organ contracting and pulling tooth out of crypt.

Dates of Eruption and Formation

	Erupt.	Roots complete.	Absorption begins.
Central	7 months.	3 years.	$4\frac{1}{2}$ years.
Lateral	9 „	3 „	$4\frac{1}{2}$ „
Canine	18 „	5 „	$6\frac{1}{2}$ „
1st molar	12 „	4 „	$5\frac{1}{2}$ „
2nd molar	24 „	5 „	$6\frac{1}{2}$ „

Erupt early in—

a. Syphilitic children (some may erupt before birth).

b. Tuberculous children.

Erupt late in—

a. Rickety children.

b. Unfavourable hygienic surroundings.

c. Cretinism.

Normal Signs of Approaching Dentition

1. Increased flow of saliva.
2. Tendency of child to bite.
3. Gums tense and shining.
4. Mouth hot.

Complications of Eruption of Temporary Dentition

Local:—

1. Acute inflammation of gums, especially over tooth.
2. Ulceration; if extensive, called "Odontitis Infantum."
3. Cyst over erupting tooth, especially in rickety children.

Treatment—

Lance gums.
Astringent to ulcer.
Purge.
Potassium chlorate gr. j. t.d.s.
Incise cyst.

General:—

1. *Nervous System—*

Restlessness.
Sleeplessness.
Paralysis.
Convulsions.

Treatment—

Warm mustard bath.
Purge.
Brandy (few drops every half-hour).

2. *Respiratory System—*

Cough.
Bronchitis.

3. *Alimentary System—*

Diarrhœa.
Constipation.
Vomiting.

4. *Integumentary System*—
   Erythema.
   Eczema.
   Herpes.
   Impetigo.
5. *Genito-urinary System*—
   Increased or diminished urine.
   Urethritis.
6. *Coryza.*

It may be noted that "every ill that flesh is heir to" in a baby has been attributed to teething. But with the advance in the scientific feeding of infants, there is much evidence to prove that many of the affections that have been put down to teething are really largely due to errors of diet, the teething process only acting possibly as a predisposing factor in these cases by the pain and consequent exhaustion, causing a reduction in the general vitality of the child. The nervous complications are certainly more likely to be directly due to the teething, by the stimulus of the erupting tooth acting reflexly upon the nervous system.

Absorption of Roots of Temporary Teeth

Dates of absorption given above.

Process.

Root at or near its end becomes excavated by cup-shaped depressions (*Howship's Lacunæ*), which deepen and coalesce, and thus root is eaten away.

Generally begins on side next the successional tooth, but not always—hence independent of pressure.

Cementum is attacked first, then dentine, and sometimes then enamel.

That part of dentine next pulp is more resistant than other parts, and hence often persists for some time as a hollow column.

Absorption depends upon the integrity of the pulp. If dead, root is only slightly absorbed or not at all. Hence so-called "necrosis of roots of temporary teeth" and protrusion through the gum.

Rate of absorption not constant. May stop for a time and give rise to deposition of cementum, and these processes may go on alternating, the absorption finally exceeding the deposition.

Theories of Absorption.

1. *Absorbent Organ of Tomes.*

Very vascular soft tissue applied to excavations caused by absorption. Surface covered with large cells, somewhat like myeloid or giant cells, each of which fits into an indentation of excavated surface.

3 layers of organ—

a. Large giant cells.
b. Ordinary nucleated cells.
c. Fibrous tissue.

Action of organ. Theories—

a. Cells put forth amœboid processes.
b. Cells secrete an acid fluid.
c. Inflammatory process.

2. *Pressure Theory.*

Compare absorption of lower 2nd molar by advancing 3rd molar. Really independent of pressure, as given above.

3. *Cannibal Theory.*

Eaten up or assimilated by advancing tooth. This cannot apply to absorption of permanent teeth.

B. ERUPTION OF THE PERMANENT TEETH

Analogous to that of milk teeth, an orifice very much larger than crown of tooth being opened up. Hence slightest force directs the rising crown ; *e.g.* :—

a. Fragment of root of temporary tooth.
b. Action of lips and tongue.

Temporary teeth stood vertically, but permanent teeth stand obliquely forwards, thus making room for canine, which was out of line during development.

When crown has erupted and reformed bone is enclosing neck, little of root is complete, the *short* and *widely open* root occupies the whole depth of socket, and so as root gets longer its socket is deepened by additions to its *free edges*, and not by the root burrowing deeper into the alveolar process. Thus the sockets grow up with and are moulded round the teeth as they elongate, and therefore the whole of the alveolar process is subservient to the position of the teeth.

A symmetrical arch is moulded by the lips and tongue, giving constant and equal pressure on each side.

Dates of Eruption and Formation

	Erupt.	Roots complete.
Central incisor . .	7 years.	10 years.
Lateral ,, . .	8 ,,	11 ,,
Canine . . .	12 ,,	13 ,,
First premolar . .	10 ,,	12 ,,
Second ,, . .	11 ,,	12 ,,
First molar . .	6 ,,	9 ,,
Second ,, . .	13 ,,	16 ,,
Third ,, .	18–24 ,,	20 ,,

RETENTION OF TEMPORARY TEETH WITH PERMANENT TEETH

In order of frequency:—

Upper canines.
Second lower molars.
Second upper molars.
Lower incisors.

May remain till quite late in life.

Results:—

May remain at original level.
May rise to occlusion.
Gum may grow over them.
May be wedged between two permanent teeth.
Caries from wedging.
Irregularities of permanent teeth.

Treatment:—

Take skiagraph to ascertain position of successor, if unerupted.

a. If in a position to erupt, extract temporary tooth.

b. If absent or much out of position, retain temporary tooth if healthy.

CHAPTER III

IRREGULARITIES OF THE TEETH

A. Temporary Dentition.
B. Permanent Dentition.

Abnormalities in—

1. Size.
2. Number.
3. Structure.
4. Position.

A. TEMPORARY DENTITION

1. Abnormalities in Size :—

Upper canine.
2nd temporary molar. } Often larger.

2. Abnormalities in Number :—

Supernumeraries and supplemental teeth.
Usually in incisor region.
Cases on record of total absence.

3. Abnormalities in Structure :—

Additional cusps.
Additional roots—2nd molars and lower canine.
Honey-combed (rare).
Geminated. Most common in lower incisors and canines. (See below.)

4. Abnormalities in Position :—

Teeth—overlapping and twisting.
Jaws—protruding and open bite.

B. PERMANENT DENTITION

1. Abnormalities in Size

Abnormally *large* teeth are generally :—

Upper central.
Lower 2nd premolar.
Lower 2nd molar.

Abnormally *small* teeth are generally :—

Upper lateral.
Upper 3rd molar.

2. Abnormalities in Number

a. Supernumeraries.
b. Supplementals.
c. Absence of some or all.

a. Supernumerary Teeth

Most common in—

(i) Upper incisor region.
(ii) Upper 3rd molar region.

Characteristics :—

(i) Generally single root.
(ii) Enamel ends straight round middle of tooth.
(iii) Any form from simple conical tooth to small molar. —With the latter the cusps and roots are rather convergent than divergent.

Theories of Formation :—

(i) *Sutton.*—Dichotomy—*i.e.* complete unequal division of tooth germ.
(ii) *Mallassez.*—From masses of epithelium or epithelial pearls.
(iii) *Atavism.*

b. Supplemental Teeth

Most common in—

(i) Upper and lower lateral incisor region.
(ii) Premolar region.

Characteristic.

Simulate the teeth next to which they stand.

Theory of Formation.

Dichotomy, with complete equal division of tooth germ.

c. Absence of Teeth

Most common in—

(i) Upper lateral incisor region.
(ii) Lower 2nd premolar region.
(iii) Upper 3rd molar region.

Occasionally seen—

(i) Absence of several teeth.
(ii) Absence of all teeth.

These often associated with other integumentary abnormalities.

3. Abnormalities in Structure

Forms :—

a. Syphilitic teeth.
b. Stomatitic teeth.
c. Geminated teeth.
d. Enamel nodules.

a. Syphilitic Teeth

Found in people the subjects of *congenital* syphilis.

Characteristics :—

(i) Narrowing of tooth from gum to cutting edge.

(ii) Semilunar notch at cutting edge of upper incisors in adult life, which on eruption is occupied by frail enamel, which soon wears away, leaving the notch. Laterals are not always affected.

(iii) Margins of upper incisors are rounded and ill-defined, and distal edges turned outwards.

(iv) Peg-top shape to lower incisors.

(v) All teeth small, and spaced as a rule.

(vi) Implantation very weak.

(vii) Dome-shaped 1st molars with dwarfed, rounded cusps.

(viii) Enamel, normal in colour and thickness.

(ix) Lime salts deficient—hence soft and prone to caries.

Temporary Dentition.

Not as a rule affected. May be small and dusky hue, indicating bad calcification.

Coles reports a case where incisors had characteristic markings, as in Permanent Dentition.

Causation.

Very little understood.

Hutchinson says it is due to specific stomatitis occurring about time of or just before birth.

This would affect all teeth developing at that time, but this apparently is not found.

Dentine is much more affected than *enamel*.

b. Stomatitic Teeth

Also called—

Hypoplastic teeth.
Honeycombed „
Mercurial „
Rocky „
Craggy „

Teeth affected.

Generally—
Incisors.
Canine (tip).
1st molar.
Sometimes—
Premolars.
2nd molars.
All teeth.

Characteristics :—

(i) Lines of dots or pits transversely across teeth, the number varying.
(ii) Shallow grooves transversely across teeth may be found.
(iii) Molars—the contoured portion presents a marked shoulder or rim, and cusps are merely spinous projections.
(iv) Brown stain when enamel is worn.

Microscopical appearances :—

(i) Enamel thin or absent in grooves and pits.
(ii) Prisms and striæ of Retzius well marked.
(iii) Prisms may be granular.
(iv) Some parts—enamel is homogeneous brown mass.
(v) Interglobular spaces well marked.

Causes :—

(i) Any illness that interferes with calcification, while it is in progress.

(ii) Especially exanthematous fevers, as those are the commonest severe illnesses from which a child suffers.

(ii) Bad feeding at same periods.

(iii) Hutchinson says it is due to mercury given during first two years of life.

c. Geminated Teeth

Forms :—

1. True or Developmental Gemination.
2. False or Pathological Gemination.

1. True Gemination.

Causes :—

(i) *Close apposition and fusion of tooth germs*—giving rise to gemination of two normal teeth of the arch.

(ii) *Incomplete dichotomy*—giving rise to gemination of one normal tooth with a supernumerary or supplemental.

Varieties in order of frequency :—

Fusion of two incisors.
„ incisor and supernumerary.
„ molar and supernumerary.
„ lateral and canine.
„ two molars.
„ canine and premolar. } Very rare.
„ two premolars. } Very rare.
„ premolar and molar. } Very rare.

Union :—

(i) *By whole length of tooth* (most common).
There is then continuity of enamel, dentine and cementum, with either one or two pulp chambers.

(ii) *By crowns only.*
There is then continuity of enamel and dentine, with either one or two pulp chambers.

(iii) *By roots only.*
There is then continuity of dentine and cementum, with either one or two pulp chambers.

2. False Gemination.

Cause.

Chronic inflammation of periodontal membrane, leading to *organisation*, *i.e.* productive periodontitis.

Varieties.

Any adjacent teeth may be united.
Especially common in molars.

Union.

By cementum only.

d. Enamel Nodules

Enamel excrescences on roots of teeth.

Site.

Generally upon molars between roots.
May be found occasionally on single-rooted tooth.

Structure.

Generally core of dentine connected with that of the root, covered with thick enamel.
Occasionally small pulp cavity found.
May be enamel only, and outside the cementum.

Origin.

If enamel only—formed by portion of "*sheath of Hertwig*" developing enamel.

If with dentine core—formed by dichotomy of tooth germ. Therefore transition from enamel nodules to supernumerary teeth is only one of degree.

4. Abnormalities in Position

Types described :—

a. Superior Protrusion.
b. Inferior Protrusion.
c. Open Bite.
d. Misplaced Lower 3rd Molar.

a. Superior Protrusion

Causes :—

(i) *Connected with Maxilla.*

Excessive development—
Alveolar process.
Whole bone.
Overcrowding of teeth.
Supernumerary teeth.
Narrow or small arch.
Large teeth.

(ii) *Connected with Mandible.*

Deficient development.
Crowding of lower incisors—fan-shape.
Retardation of eruption of molars, causing mastication to take place on front teeth.
Early extraction of first permanent molars doing the same as above.

(iii) *Connected with Soft Parts.*

Attached frænum.
Short upper lip, and hence absence of anterior support to upper incisors.

(iv) *Habits of Children.*

Thumb, finger, toe, lip, and tongue sucking.
Rubber teat.

(v) *Obstructed Respiration.*

Theories of action :—

a. Tension at sides of mouth by being open.

β. Absence of tongue support to upper.

γ. Under development of nasal fossæ ; their function being lost from want of use. Hence floor of nose, *i.e.* palate, is higher and narrower than normal, and so sides are drawn together and anterior part protruded.

δ. Atmospheric pressure greater in the mouth than in the nose with inspiration. Hence palate forced up.

(vi) *Fault in Temporo-maxillary Articulation.*

Causing posterior bite (?).

Treatment.

Note :—

Facial contour.
Profile.
To determine which parts of arches at fault, and therefore which, if possible, to be corrected.

Observe :—

Anterior occlusion.
Class A. Plenty of room between upper and lower incisors, for the former to be drawn in.
Class B. Lower incisors biting in such a situation that the upper ones would be bitten out again if merely drawn in

Class A.

Methods available :—

a. **Retract upper incisors,** when—

(i) Normal molar occlusion.

(ii) Only slight distal occlusion.

If further space in upper necessary for this treatment, decide "expansion" or "extraction" according to :—

(i) Facial contour—expansion broadens face.

(ii) Amount of room wanted—expansion less than extraction.

(iii) Time at patient's disposal — expansion takes longer.

(iv) Direction of roots—expansion may tilt teeth too much.

With *Expansion* — generally necessary to expand lower jaw as well.

Appliances used :—

(i) Coffin's spring plate.

(ii) Read's screw plate.

(iii) Badcock's screw plate.

(iv) Angle's arch.

(v) Jack-screw.

With *Extraction,* be guided by :—

(i) Which teeth carious.

(ii) Amount of space wanted.

(iii) Distance from irregularity.

(iv) Possibility of space left after retraction.

To retract incisors.

Appliances used :—

(i) Angle's wire arch or modification, with or without skull-cap.

(ii) Badcock's screw plate and spring wire arch.

(iii) Rubber band with plate.
(iv) Wire bows on plate.
(v) T-piece on plate, &c.

b. **Jump the bite,** when—
Distal molar occlusion is great.

Appliances used :—
(i) Reciprocal traction with rubber bands. (Baker anchorage, &c.)
(ii) Anterior inclined plane.

Means of retention :—
(i) Interlocking wedges on buccal surface of molars.
(ii) Linked chain anchorage.
(iii) Inclined plane.

Class B.—Preliminary Treatment.
Methods available :—
a. Insert bite plate in upper, leaving molars free to rise and occlude, and which also tends to depress lower incisors.
b. Extract one of lower centrals and insert bite plate to depress the others.
c. Extract unerupted premolars, if case seen early enough, and insert bite plate.
d. Grind down and polish lower incisors, if only slight amount necessary.

After any of these methods of preliminary treatment, the case is resolved into *Class A*, and is then further treated as such.

b. Inferior Protrusion

Causes :—
(i) *Connected with Mandible.*
Excessive development.
Oblique ramus, causing horizontal part to be pushed forward.

(ii) *Connected with Maxilla.*
Deficient development.
Tardy eruption of upper incisors.
Instanding upper incisors, from late retention of temporary incisors.

(iii) *Habits of Children.*
Hooking fingers over lower incisors.
Protruding lower jaw.

Treatment :—

(i) Retract lower incisors, extracting premolars if necessary.
(ii) Expand upper jaw.
(iii) Separate all upper teeth (Kingsley).
(iv) Skull-cap and chin bandage, if due to habit.

c. Open Bite

Causes :—

(i) Elongation of molars :—
α. Keeping mouth open—
Obstructed respiration.
Cicatrix.
β. Not capped on regulation plate.
(ii) Congenital syphilis—
Small incisors.
Small alveolar process.
(iii) Thumb and finger sucking, &c.
(iv) Rubber teat.
(v) Imperfect development of maxilla and premaxilla.
(vi) Small or oblique ramus.
(vii) After fracture—malunion.
(viii) Hypertrophied tongue.
(ix) Changes in joint—
Dislocation.
Osteo-arthritis.
(x) Scar tissue in front part of neck in child, by contraction, moulding anterior part of mandible downwards.

Treatment :—

(i) Extraction of occluding teeth.
(ii) Grind down occluding teeth; several sittings extending over several months.
(iii) Chin bandage and skull-cap to force occluding teeth into sockets.
(iv) Elongation of upper incisors by banding and pulling to wire bow.
(v) Treat obstructed respiration.

d. Misplaced Lower Third Molar

Directions :—

(i) Obliquely forwards against second molar.
(ii) Directly forwards against second molar.
} "Impacted."
(iii) Inwards.
(iv) Upwards (may reach sigmoid notch).
(v) Downwards (inverted).

Symptoms and Results.

(i) Pain—
  α. In tooth—throbbing, jumping, or continuous.
  β. In gum—worse on eating.
  γ. In another tooth—referred.
  δ. In second molar—
    From pressure.
    From absorption.
  ε. Neuralgia—especially in ear.
(ii) Swelling of gum—painful to bite on.
(iii) Ulceration of gum.
(iv) Suppuration in tissues surrounding crown of tooth leading to—
  α. Swelling of face.
  β. Swelling at angle.
  γ. Swelling of neck.

δ. Swelling of fauces.
ε. Swelling of pharynx.
Any of which swellings may suppurate.
(v) Lymphatic glands in neck swollen.
(vi) Trismus.
(vii) Constitutional symptoms—
α. Pyrexia.
β. Rapid pulse.
γ. Malaise.
δ. Constipation, &c.

Treatment :—

(i) Take skiagraph, if position uncertain.
(ii) Lance gum by crucial incision, if plenty of room and gum only involved.
(iii) Extract offending tooth with straight elevator, if symptoms warrant.
(iv) Extract second molar, if impossible to remove third—
α. As preliminary to removal of latter, if suppurating ;
β. To relieve pressure.

CHAPTER IV

BACTERIOLOGY OF THE MOUTH

Bacteria, according to shape and arrangement are thus classified :—

1. Bacilli—rods.
2. Cocci—round bodies :—
   a. Streptococci—chains.
   b. Staphylococci—little packets.
   c. Diplococci—pairs.
   d. Sarcinæ—four in a square.
3. Spirilla—spirals.
4. Leptothrix—thread forms.
5. Streptothrix—fine threads with lateral branches.

Mouth Bacteria

In the human mouth Miller found and cultivated nearly 100 different forms—some pathogenic (disease giving) and some non-pathogenic.

But six forms he described as almost constant, and all having the same peculiarity—that no culture medium was found on which they would grow. These are :—

1. *Leptothrix innominata.* Found in soft white deposit on teeth. Stains faint yellow with iodine.
2. *Bacillus buccalis maximus.* Rods and threads, 2–10 μ long and 1–1·3 μ wide. Stains deep violet with iodine.

3. *Leptothrix buccalis maximus.* Same form as above, but no iodine reaction.

4. *Iodococcus vaginatus.* Common in unclean mouths. Chains of cells in sheath. Cells stain violet and sheath very little with iodine.

5. *Spirillum sputigeneum.* Found on inflamed gums. Form is like a comma.

6. *Spirochete dentium.* Found on inflamed gums. Long spirals, 8–25 μ long.

To these may be added :—

7. *Leptothrix Racemosa* (Vicentini). Found in soft white deposit. Felted masses of entwined threads, with "fruitful heads," *i.e.* spore-like areas.

8. *Strepothrix buccalis* (Goadby). Found in soft white deposit, and pyorrhœa. Filamentous forms with lateral branches, whose ends are often club-shaped.

Amongst the many others found in the mouth the following ordinary pathological bacteria are frequently observed :—

1. Pneumococcus.
2. Diphtheria bacillus (Klebs-Lœffler).
3. Bacillus Coli Commune.
4. Streptococcus pyogenes.
5. Streptococcus brevis.
6. Bacillus pyocyaneus.
7. Micrococcus tetragenous.
8. Tubercle bacillus.

Miller estimated in one very unclean mouth as many as 1,140,000,000 organisms.

Bacteriology of Caries

Some of mouth bacteria can *ferment* grape sugar into *lactic acid.*

Others of mouth bacteria can *peptonise, i.e.* render soluble, albumen, gelatin, and fibrin.

Hence *present view of cause of Caries:—*

> Certain mouth bacteria are capable of generating an acid which can dissolve the salts out of the tooth substance; while others are capable of dissolving the decalcified matrix—*i.e.* the organic portion remaining after the salts have gone.

Acid forming Bacteria.

Streptococcus brevis. Staphylococcus necrodentalis. Staphylococcus albus.	In deep layers.
Streptococcus brevis. Sarcina lutea. Sarcina aurantiosa. Sarcina alba. Staphylococcus albus. Staphylococcus aureus.	In superficial layers.

Bacteria capable of liquefying dentine (decalcified):—

Bacillus mesentericus ruber. „ „ vulgatus. „ „ fuscus. „ furvus. „ gingivæ pyogenes. „ liquefacieus flourescens motilis. „ subtilis. Proteus Zeukeri. Bacillus plexiformis.	All in superficial layers.

Bacteriology of Pulpitis

Forms of Bacteria found (Miller).

1. Cocci and diplococci (pathogenic).
2. Bacilli—curved and often forming threads.
3. Short rods with bipolar staining.

Individual Bacteria described.

1. Streptococcus (either pathogenic or non-pathogenic).
2. Staphylococcus albus and aureus.
3. Micrococcus tetragenous.
4. Sarcinæ. (Zierler.)
5. Bacillus gangrenæ pulpæ. (Arkövy.)
6. Bacillus mesentericus vulgatus. } Goadby and
7. „ „ ruber. } Sieberth.
8. „ „ fuscus }

Goadby holds that 5 and 8 are identical.

Bacteriology of Alveolar Abscess

Cocci mostly found, but not as a rule the common pus cocci (staphylococcus aureus), which are rarely found.

Bacteria commonly found.

1. Staphylococcus viscosus. (Goadby.)
2. Staphylococcus albus.
3. Bacillus gangrenæ pulpæ. (Arkövy.)
4. Bacilli of mesenteric group.

Bacteria occasionally found.

1. Micrococcus tetragenous.
2. Streptococci.
3. Bacillus Coli.
4. Blastomycetes.
5. Staphylococcus aureus.

Bacteriology of Pyorrhœa Alveolaris

Forms of Bacteria found (Goadby).

1. Cocci—generally diplococci.
2. Bacilli—
   a. Thick and often jointed.
   b. Thick, with pointed ends.
   c. Fine, with irregular banded markings.
   d. Masses, with some jointed in chains.
3. Spirilla and spirochæte.
4. Yeast forms—various.
5. Streptothrix—with well marked club ends.

Ordinary pus cocci (staphylococci), rarely found.

None of the organisms found have yet been shown to be *especially* related to the disease.

CHAPTER V

CARIES

Disintegration of tooth substance extending from without inwards towards the pulp, caused by external agencies.

Macroscopical Appearances

Enamel.

If starting in a crevice—a *dark* spot.
If starting on a plain surface—a *white* spot.
Starts usually by enamel losing its translucency, whereby a whitish spot appears, becoming darker if process is slow, and remaining white if rapid.
Surface roughened and disintegrated by the disorganised enamel prisms being washed away.
If undermined by caries it appears bluish-white.

Dentine.

This is now reached.
It is first softened and then disintegrated and washed away, forming a cavity; rapidity of process depending upon:—

a. Structure of tooth.
b. Virulence of micro-organisms.
c. Intensity of ferments.

Rapid or Spreading Caries (*Caries acuta*).—If calcification bad, or many interglobular spaces, caries spreads rapidly sideways, undermining the enamel, which is then attacked from the dentinal side, and is called "secondary enamel decay" (Miller).

This kind of caries is also generally moist (*Caries humida*), and light yellow or white (*Caries alba*).

Slow or Penetrating Caries (*Caries chronica*).—If calcification is good, caries only extends in directions of dentinal tubes. The shape of the cavity is then conical, with apex towards pulp.

This kind of caries is also generally dry (*Caries sicca*), and brown or black (*Caries pigmenta*), which is probably produced by chromogenic bacteria.

Cementum.

Caries may attack any portion exposed to mouth.

At first softened and later wide, shallow cavities formed.

May be deep cavity at angle between roots.

Translucent Zone.

A more or less translucent condition of the dentine, preceding the advance of the caries, and found between the caries and the pulp cavity.

Forms—

a. Regular zone.
b. Irregular cone—apex to pulp.
c. Stripes and spots.

Resembles roots of teeth of aged persons.

Not a phenomenon of caries alone, because it is seen when only enamel is worn.

Has never been produced artificially.

Due to—

Some change that renders the refractive indices of matrix, sheaths, and fibrils very nearly alike, *i.e.*—

a. Calcification of contents of tubes.
b. Partial decalcification of matrix.

Arguments in Favour of Calcification of Contents of Tubes:—

1. Diameter of tubes is lessened in translucent zone, and not permeated by Charters White's celloidin stain.
2. Takes stain with difficulty, whereas even very slightly decalcified dentine stains readily.
3. Cannot be produced artificially, which should be easy.
4. Not found in carious, pulpless teeth.
5. Not found in natural teeth worn as artificial substitutes, when caries supervenes.
6. Occurs under worn enamel.
7. Decalcification advances in a regular line, whereas this may be very irregular.

Microscopical Appearances

Enamel.

Felt-like mass of micro-organisms on surface.
Interprismatic substance dissolving away from surface inwards.
Prisms therefore loosened, partially separated, and unduly distinct.
Prisms may appear granular and pigmented if process slow.
Bacteria between prisms.

Dentine.

Mass of debris on surface.

Irregular spaces (*liquefaction foci*) in dentine, near surface, full of broken-down pieces of dentine and micro-organisms, which have entered the dentinal tubes, bulged them, and finally broken through them by liquefaction of the matrix, which has been previously decalcified by acids that passed down the tubes.

Further in, dentinal tubes full of micro-organisms and bulged, but still intact. Also, tubes normal in calibre with micro-organisms.

These latter, and also many other tubes that contain no micro-organisms, present the "*tobacco-pipe*" appearance (seen on transverse section of tubes).

This appearance is due to the partial decalcification of that portion of the matrix immediately surrounding the "sheath of Neüman," thereby rendering the sheath unduly distinct.

In *translucent zone*—rod-shaped elements and rows of shining granules in tubes, which may be looked upon as consolidated portions of fibrils.

Cementum.

Destruction takes place exactly in same manner as in dentine.

Micro-organisms penetrate principally along Sharpey's fibres, but also along canaliculi, and form liquefaction foci.

Causes of Caries

1. Predisposing—
   a. Structural.
   b. Local conditions.
   c. General conditions.
2. Exciting.

1. **Predisposing.**

a. Structural Defects.

(i) Deficiency of lime salts:

Females.
Rickets.
Temporary teeth.

(ii) Presence of pits and crevices.

(iii) Increased number of interglobular spaces.

(iv) Passage of dentinal tubes into enamel—communicating with enamel spindles.

b. Local Conditions in the Mouth.

(i) Irregularities of teeth, being inaccessible for cleansing.

(ii) Crowded arch for same reason.

(iii) Want of cleansing.

(iv) Wearing of dentures—collecting debris.

(v) Recession of gum.

(vi) Class of food:—

Carbohydrates especially deleterious.

Proteids and fats have little or no effect, *e.g.* Esquimaux—fat eaters; no caries.

Foods from which fibrous material has been eliminated, *e.g.*:—

Roller-milled flour.
All cooked food.

Fibrous material when retained tends to cleanse teeth during mastication.

Sticky foods, *e.g.*:—

Cooked starchy foods.
Sweets, &c.

All tend to cling, and may be fermented into acid *in situ* on teeth.

(vii) Abnormal secretions of mouth :—

Acid saliva in dyspeptics.
Sticky mucoid saliva in—
Pregnancy.
Fevers.
Most illnesses.

(viii) Medicines :—

Mineral acids.
Iron salts.

c. General Conditions.

Besides those mentioned incidentally above under "Local Conditions" :—

(i) Hereditary influence.

(ii) Race.

(iii) Civilisation.

(iv) Occupation :—

Millers. } Flour.
Bakers. }
Fur-dyers, &c. :—
Acid fumes.

(v) Artificial feeding of infants.

(vi) Children brought up in districts poor in lime salts in food and water.

Immunity.

Besides all the above predisposing causes there is still some further undiscovered factor which is not understood at all, to explain the facts that sometimes perfect teeth under *apparently* perfect conditions will decay, and also that sometimes badly formed teeth, even under what are considered bad conditions, will escape caries for many years. Miller looked

upon this immunity as due to some bacteriacidal property of the saliva, which, however, he could not determine. Also, it may be that certain saliva has the property of attracting leucocytes in the presence of bacteria.

2. **Exciting Causes** (in historical order).

a. Inflammatory Theory (Abbot).

That caries was set up by inflammation of living tooth substance internally.

Evidence against:—

(i) If right, filling would promote caries.
(ii) No caries without breach of enamel.
(iii) Caries goes on in dead tooth.

b. Electrical Theory (Bridgeman).

Crown—positive pole.
Root—negative pole.
Saliva—electrolyte.
} Action set up by abnormal saliva.

c. Chemical Theory.

Due to acids of mouth (see below).

d. Parasitic Theory.

Due to micro-organisms alone.

e. Chemico-parasitic Theory (present accepted theory).

Due to action of acids of mouth and micro-organisms.

Decalcification by acids.

Dissolution or peptonising of the remaining organic substance by bacteria acting in an acid medium.

Origin of Acids of Mouth:—

a. By fermentation.
b. Introduced with food.
c. Regurgitated from stomach.
d. Secreted from mucous glands.

a. By Fermentation.

Partly by ptyalin of saliva.

Partly by bacteria themselves.

Sugar and other carbohydrates ferment into—

Lactic acid.
Butyric acid.
Propionic acid.
Valeric acid.

Albumen *may* ferment into—

Butyric acid.
Valeric acid.

For fermentation process, see below.

b. Introduced with Food.

Fruit, vegetables, drinks, &c., contain—

Citric acid.
Malic acid.
Oxalic acid.
Acid oxalates.
Acetic acid.
Tartaric acid.
Acid tartrates.
Alum.
Chloride of iron.
Perchloride of iron.
Sulphate of iron.

All of which have a greater or less dissolving action upon the dental tissues.

c. Regurgitated from Stomach.

Small quantities of—

Lactic acid.

Hydrochloric acid.

d. Secreted from Mucous Glands.

May be *very* small quantities in certain affections.

Fermentation of Carbohydrates.

$(C_6H_{10}O_5)_n$	$C_{12}H_{22}O_{11}$	$C_6H_{12}O_6$
Starch	Maltose	Dextrose
Dextrin	Lactose	Levulose
Cellulose	Cane sugar	Galactose

Action of Ptyalin.

Cellulose (coating of starch grains) not acted upon, and therefore uncooked starch is unaltered by saliva.

$$10(C_6H_{10}O_5)_n + 4nH_2O = 4n(C_{12}H_{22}O_{11}) + (C_6H_{10}O_5)_n + (C_6H_{10}O_5)n.$$

Starch. → maltose + achroo-dextrin + erythro-dextrin.

These gradually change to maltose.

Action of Bacteria.

$$C_{12}H_{22}O_{11} + H_2O = C_6H_{12}O_6 + C_6H_{12}O_6$$

(i) Maltose = dextrose + dextrose.

(ii) Lactose = dextrose + galactose.

(iii) Cane sugar = dextrose + levulose.

$$C_6H_{12}O_6 = 2C_3H_6O_3$$

Dextrose, Levulose, Galactose } = Lactic acid.

Artificial Production of Caries.

All phenomena can be reproduced except translucent zone.

Method—

Keep teeth or portions of teeth in mixture of *bread* and *saliva* for three months at 98° F., and renew it every two or three weeks. Otherwise putrefaction sets in and the mixture becomes alkaline, and then bacteria cannot act.

Resultant caries is *white*, but by allowing putrefaction at certain stages or by exposure to air or various coloured articles of food, all sorts of colouration appear.

In the mixture—

Bread is to supply the carbohydrates to ferment.

Saliva is to supply bacteria, ptyalin, and moisture.

CHAPTER VI

DISEASES OF THE PULP

In describing the inflammations of the Pulp, it is as well to point out clearly that there are no hard-and-fast lines between the different types of inflammation described, but that they are only thus classified for purposes of description. The different kinds merge into one another, and it is quite impossible to determine where one ends and another begins.

The types of inflammation of the pulp described below are :—

1. *Hyperæmia*, or mild inflammation, extending over short period only, or intermittent with healthy intervals.
2. *Acute inflammation*, or severe condition, extending over short period.
3. *Chronic inflammation*, or variable condition, extending over long period.

HYPERÆMIA OF PULP (*Pulpitis*)

Causes :—

1. Thermal changes in mouth.
2. Advancing caries, erosion, attrition, abrasion.
3. Exposure of cementum.
4. Sweet and acid substances acting through 2 and 3.
5. Filling too near pulp.
6. Fracture of tooth.
7. Incautious use of As_2O_3, $ZnCl_2$, carbolic acid, &c.
8. Susceptibility on part of patient.

Symptoms :—

1. Intermittent pain—
   Paroxysmal.
   Jumping.
2. Thermal shocks.
3. May be slightly painful to mastication (when hyperæmia has extended to periodontal membrane).

Treatment :—

1. Remove cause.
2. Oil of cloves and temporary filling (oxysulphate).
3. Non-conducting filling.
4. Counter-irritant to gum.
5. Removal of pulp, if persistent.

Microscopical Appearances :—

1. Enlarged and tortuous vessels.
2. Great increase of pulp cells due to proliferation of pre-existing cells and to escape of leucocytes.
3. Increase of odontoblasts, which are arranging themselves in "sheaves" near region of irritation.
4. Cylindrical calco-globulin formations in neighbourhood of vessels.

Pathological Results :—

1. If cause removed—
   a. Generally ends in resolution.
   b. More rarely passes on to acute or chronic inflammation.
2. If cause persists—
   Passes on to acute or chronic inflammation.

ACUTE INFLAMMATION OF PULP

(*Acute Pulpitis*)

Causes :—

Same set of causes as for Hyperæmia (see above), only in a more pronounced degree or extending over longer period, especially if the pulp has

become exposed, allowing irritation from foreign matter.

Also As_2O_3 for devitalising.

Symptoms :—

1. Sharp throbbing pain, worse in recumbent position and more or less continuous.
2. Thermal changes increase pain, though cold *may* relieve in early stage.
3. If exposure of pulp—
   Wool in cavity causes pain.
   Jet of warm water causes pain.
4. Tooth may be little tender to pressure, if inflammation extended to periosteum.

Treatment :—

1. Removal of pulp—
   Anæsthetise with cocaine.
   Devitalise with arsenic.
2. If in early stage, may be saved by :—
   a. Dressings with—
      Obtundents.
      Astringents.
      Sedatives.
   b. Capping with a non-irritant and non-conducting filling. This method should only be adopted in a young and perfectly healthy subject, otherwise degeneration and death of pulp are likely to follow. It is a doubtful procedure, even under favourable circumstances.
   c. Extract tooth if not saveable.

Microscopical Appearances :—

1. All the appearances as in Hyperæmia (see above), only more exaggerated.
2. Altered odontoblasts—shrunken or enlarged.
3. Nerve bundles lost their definite structure.

4. Minute abscesses with cells broken down, appearing as little blurred patches with broken centres in stained section.
5. Micro-organisms if exposed pulp or only thin layer of dentine covering.

Pathological Results (in the pulp):—

1. Resolution—rare.
2. Suppuration, common because of—
   a. Delicate structure.
   b. Inability to expand.
3. Passing into chronic inflammation.
4. Death *en masse* (gangrene) from strangulation of vessels at apex.

Changes during Resolution.

Cause ceases, or leucocytes overcome bacteria.
Bacteria if present and toxins absorbed by leucocytes.
Leucocytes and plasm pass back into capillaries.
Blood flow again becomes active.
New vessels formed, when some quite thrombosed, by budding off from endothelium.
Some leucocytes become fibricated and then converted into connective tissue.
Vessels return to normal size.

Changes during Suppuration.

Bacteria and toxins overcome leucocytes, which die.
They liberate proteolytic ferment, which dissolves cells and tissues.
This occurs in small patches, especially near exposure. These extend and coalesce, and finally whole pulp liquefies.

CHRONIC INFLAMMATION OF PULP

(*Chronic Pulpitis*)

Causes:—

Following Hyperæmia or acute inflammation of pulp from any of their causes as given above.

Symptoms :—

Very variable.
Generally pain of intermittent and neuralgic character, but may be any degree of severity.
May be painful to mastication.
May give rise to no symptoms at all.

Treatment :—

Removal of pulp.
Removal of tooth if not saveable.

Pathological Results (in the pulp) :—

1. *Suppurative.*
   a. On surface—ulceration of pulp.
   b. Complete—death of pulp.
2. *Destructive.* Absorption of dentine of pulp cavity.
3. *Productive.*
   a. Growth of pulp into absorbed dentine.
   b. Growth of pulp into carious cavity (*Polypus*)
   c. Calcification of above growth.
4. *Degenerative.*
   a. Fibroid changes.
   b. Calcareous changes.
   c. Fatty changes.

Polypus of Pulp (*Hypertrophic Pulpitis*)

Cause :—

Chronic Pulpitis (see above).

Symptoms :—

Fleshy mass in carious cavity.
Bleeds easily.
Insensitive.

Treatment :—

If tooth saveable, remove polypus and fill or crown tooth. (Apical foramen may be too large by absorption.)

If tooth not saveable, extract. Great care must be exercised, as tooth is often very fragile on account of absorption of pulp cavity.

Pathology :—

Pulp hypertrophied—

a. In carious cavity by growth of granulation tissue.

b. In pulp chamber by absorbing surrounding dentine.

Microscopical appearances—

Granulation tissue.

Fibrous tissue.

Squamous epithelium on surface (formed by kind of skin grafting).

Abundance of vessels.

Scarcity of nerves.

May undergo—

a. Calcification.

b. Any degeneration.

Fibroid Degeneration of Pulp

Sometimes described as—

1. Reticular Atrophy. (Wedl.)
2. Areolation of Pulp. (Black.)

Microscopical Appearances.

Presents reticular network throughout pulp.

Cells, vessels, and nerves all undergoing fibrification.

Odontoblasts shrunken, converted into fibrous cords or disappeared.

Pulp cells disappeared.

Vessels fewer in number, thin walled and larger, and they have constrictions at intervals by contractions of fibrous tissue.

Nuclei of sheaths have gone, and arteries and veins appear alike.

Nerves may be fatty.

Spaces formed by contraction of fibrous tissue.

May be slight calcareous formations.

Calcareous Formations in the Pulp

1. Adventitious or secondary dentine.
2. Pulp stones.
3. General calcareous degeneration.

1. Adventitious or Secondary dentine.

Causes:—

Chronic inflammation of pulp from any of its causes.

Microscopical Appearances:—

Generally definite line of demarcation between the normal and adventitious dentine.

Abrupt bend of tubes where the two join.

First part formed is often very irregular, but becomes more regular towards pulp, if extensive.

Varies very much in arrangement of tissues—tubes, spaces, cells, calcified connective tissue fibres may all be mixed up in a hyaline matrix, or one or other of these elements may predominate. Thus the following varieties are described :—

a. Tubular.
b. Areolar.
c. Lacunar.
d. Cellular.
e. Fibrillar.

2. Pulp Stones.

Causes :—

a. Chronic inflammation of pulp.
b. Gout.
c. Old age.

Symptoms :—

Often no pain at all.
If pain, generally neuralgic.

Diagnosis :—

By process of elimination.

Macroscopical Appearances :—

Occur as varying sized calcareous masses in the body of the pulp or attached to the wall.

Microscopical Appearances :—

Generally has nucleus, which is of doubtful origin ; supposed to be some small irritant spot in the pulp, around which a barrier of calcareous material has been deposited, *e.g.* a dead cell.

Often laminated concentrically about the nucleus.

May contain tubes radiating out from centre.

May have no apparent structure at all.

3. General Calcareous Degeneration.

Causes :—

Same as for pulp stones.

Macroscopical Appearances :—

Occurs as—

a. Gritty small masses throughout pulp.
b. Trabeculæ.
c. General calcification.

Microscopical Appearances :—

Vary as with pulp stones.

CHAPTER VII

DISEASES OF THE PERIODONTAL MEMBRANE

Again it is as well to point out that there is no hard-and-fast line between the different kinds of inflammation described below, as one may at any stage pass into another.

The types of inflammation described below are :—

1. Acute local periodontitis.
2. Chronic local periodontitis.
3. General suppurative cervical periodontitis (Pyorrhœa Alveolaris).
4. General periodontitis.

ACUTE LOCAL PERIODONTITIS

i.e. Acute inflammation at the root of *one* tooth.

If running its full course, it will end in acute alveolar abscess.

But it may at any stage cease of its own accord or as the result of treatment, and then resolve altogether or pass into chronic periodontitis.

Causes :—

1. Septic matter passing through apical foramen.
2. Arsenic passing through apical foramen.
3. Extension of inflammation from pulp.
4. Blow on tooth (generally accompanied by death of pulp).

Symptoms, in order of their appearance (= Clinical History).

4 *Stages,* which rapidly pass from one to the other :—

1. *Inflammatory Stage.*

   Tooth uneasy.
   Disposition to grind teeth together, giving relief at first stage.
   Tooth raised in socket, due to engorgement of vessels.

2. *Exudation Stage.*

   Gum swollen and painful, with red margin.
   Tooth more raised in socket.
   Great pain on biting, due to exudation of lymph and leucocytes.
   Increased flow of saliva.

3. *Pus-forming Stage.*

   Great throbbing and continuous pain.
   Increased swelling of gum.
   Inability to eat.
   Loss of appetite.
   Furred tongue.
   Pyrexia.

4. *Pointing Stage.*

   Face œdematous, according to situation of bad tooth.
   Pain less, as pressure relieved by pus burrowing through bone.
   Lymphatic glands of neck swollen.
   Salivary glands may be swollen.
   Constitutional disturbance.
   Abscess points.

Duration.

Average, 7 or 8 days.

Treatment:—

Stages 1 and 2.

Clear out and dress pulp cavity.
Counter-irritants to gum.
Local depletion—
Scarification.
Leeches.

Stages 3 and 4.

If saveable.

Clear out and dress pulp cavity.
If not pointing—
Local depletion.
Hot fomentations in mouth.
Poultice with hot fig, &c.
If pointing—
Open abscess.
Rhizodontrophy—as temporary measure, if tooth too tender to manipulate further.
Purgative.
If about to point externally, cause it to open inside mouth and paint collodium outside to prevent breach of skin.

If not saveable.

Extract.

Pathological Course of Acute Alveolar Abscess:—

1. Passage of micro-organisms through apex of tooth.
2. Action of these micro-organisms on the healthy or perhaps previously inflamed periodontal membrane, leading to severe inflammation around apex.
3. Progress of this inflammation to suppuration of membrane, and pus forming, strips the membrane from the root.

N.B.—No *sac* formed when process is acute, but membrane involved breaks up into pus or forms granulation tissue.

4. Pus continuing to form makes a cavity for itself in the bone, around the apex of the tooth, by the pressure of pus formation and the inflammation absorbing the bone away. Eventually bone is absorbed right through at one spot, and then—
5. Abscess points.

Situations in which an Abscess may point:—

1. Usually through outer plate.
2. Neck of tooth, along course of root.

} All the teeth.

3. Hard palate.
4. Soft palate.

$\frac{6 \cdot 5 \cdot 2}{\quad} \quad \frac{2 \cdot 5 \cdot 6}{\quad}$

5. Nasal cavity. $\frac{\quad 1}{\quad} \quad \frac{1 \quad}{\quad}$
6. Antrum. $\frac{7 \cdot 6 \cdot 5}{\quad} \quad \frac{5 \cdot 6 \cdot 7}{\quad}$
7. Along margin of mandible on face—

$\frac{7 \cdot 6 \cdot 5 \cdot 4}{\quad} \quad \frac{4 \cdot 5 \cdot 6 \cdot 7}{\quad}$

8. Fauces.
9. Pharynx.
10. Angle.

$\frac{8}{\quad} \quad \frac{\quad 8}{\quad}$

11. Neck. $\frac{8 \cdot 7 \cdot 6}{\quad} \quad \frac{6 \cdot 7 \cdot 8}{\quad}$

Complications of Alveolar Abscess:—

1. Forced closure of jaw—by exudation of inflammatory products mechanically fixing the jaw.
2. Necrosis of jaw—especially alveolar process.
3. Cellulitis of neck.
4. Inflammation and suppuration of neighbouring lymphatic glands.
5. Empyema of antrum.

6. Sinuses on face and depressed cicatrix later.
7. Trisms.
8. Œdema glottidis.
9. Septicæmia and pyæmia.
10. Post-pharyngeal abscess.
11. Thrombosis of—
    Pterygoid Plexus.
    Cavernous Sinus.

CHRONIC LOCAL PERIODONTITIS

According to the cause, this condition may be—

1. Confined to apex.
2. Confined to neck.
3. Affecting whole root.

And either 1 or 2 may become 3 if unchecked.

Also according to virulence of irritation, it may be—

1. Non-suppurative.
2. Suppurative.

Causes :—

1. Any one of the causes of Acute Local Periodontitis.
2. Traumatic effects :—
   a. Filling left too high.
   b. Crown left too high.
   c. Too rapid separating.
   d. Too rapid regulating.
   e. Impacted third molar.
   f. Denture.
   g. Oblique contact.
   h. Blow.
   i. Biting hard substance.
   j. Gold plugging.
   k. Toothpick.
   l. Rubber dam clamp.

Loss of function 5
Chronic Periodontitis.

3. Foreign bodies—
   a. Tartar.
   b. Ligature.
   c. Elastic bands.
   d. Fish bone, &c.
   e. Toothbrush bristle.
   f. Edge of ill-fitting crown.
   g. Overhanging edge of filling.
   h. Denture clasp.
4. Perforation of side of root.
5. Tooth rising in socket from loss of antagonist.
6. Arsenic dressing leaking on to gum.

Symptoms:—

With both non-suppurative and suppurative varieties:—

1. Loosening and lengthening of tooth in socket.
2. Tenderness on percussion.
3. Dull pain.
4. Gum congested.
5. Remission and recurrence.

With suppurative variety only:—

6. Gum-boil sometimes over or near root of affected tooth; *i.e.* termination of a sinus leading down to the chronic abscess cavity around the apex. This gum-boil may fill up from time to time and then burst.
7. Pus may be continually draining down canal.
8. Pus may be oozing up round neck of tooth.
9. Deposit of "seruminal calculus" on tooth from pus, if persistent.

Treatment:—

1. Remove cause.
2. If pulp dead, clear out and dress canals.
3. Apply counter-irritants to gum.
4. Remove "seruminal calculus," if confined to neck.

5. Antiseptic mouth wash, if cervical suppuration.
6. If gum-boil present, syringe forcibly through canal, and out at sinus with carbolic acid (1 in 20).
7. If above does not cure, trephine alveolus and cut off apex.

Pathological Results (directly connected with root).

Productive :—

a. Abscess Sac.
b. Granuloma.
c. Dental Cyst.
d. Exostosis and Inostosis.

Destructive :—

e. Absorption of root.

a. **Abscess Sac.**

Origin :—

When suppuration occurs on the inner surface of the Periodontal Membrane, if remaining chronic, it may gradually expand the membrane and absorb the bone, and thereby form a bag or sac, which communicates with the apical foramen and sometimes with the surface, by the pus which it contains burrowing through the bone and gum.

The inner surface of sac is lined with granulation tissue, and as time goes on its wall thickens and becomes more fibrous, and the pus from time to time discharges down the canal or through the sinus.

The sac is generally pyriform, but may be any shape. It often remains attached to the apex on removal of tooth.

b. **Granuloma.**

Origin :—

This is a mass of granulation tissue formed from the Periodontal Membrane and possibly partly from the bone. It may be formed primarily without any suppuration, but more frequently as the result of long-standing suppuration, where the pus has continually found an exit through a sinus or down the canal.

Sometimes small aggregations of epithelial cells are found in the substance of the granuloma. Occasionally these are seen in large numbers, and they are then called *Epithelial Root Tumours.*

c. **Dental Cyst.**

Origin :—

This is formed from one of the aggregations of epithelial cells frequently found in the Periodontal Membrane, especially in the neighbourhood of the apex, and which are the remains of the epithelial sheath of Hertwig. These epithelial cells are stimulated to growth by some chemical or bacterial irritant that passes through the apical foramen of a dead tooth, but which is insufficient to give rise to suppuration. They proliferate and form first an *Epithelial Root Tumour*, as given above, and then, as the peripheral cells are proliferating, the central ones degenerate and break down into fluid. This process is progressive, and eventually a large cyst is formed, which hollows out the jaw by absorbing the bone from the inside, and at the same time bone is deposited in rather less quantity on the outside, and so the appearance of expansion of the jaw is obtained.

Symptoms:—

Painless as a rule, or slight neuralgia.

May be numbness (interference with nerve trunk).

May affect either jaw.

Generally connected with permanent teeth.

Very rarely connected with temporary teeth.

May occur at any age.

Always in connection with *septic tooth* (which may, however, have been removed previously).

Smooth regular swelling.

Expands outer plate of alveolus most.

Progressive slow growth—anything from 6 months to 2 years to cause any disfigurement.

No sign of inflammation.

Hard to the touch, if bone not absorbed away.

Egg-shell cracking, if bone thin.

Fluctuation if bone absorbed.

Diagnosis:—

See below under "Swellings of Jaws."

Contents:—

Viscid, transparent fluid, containing serum-albumen, serum-globulin and crystals of cholesterin (cholesterin probably formed by degeneration of epithelial cells).

Microscopical Appearances:—

Cyst wall lined with epithelium of variable thickness and character.

Rarely epithelium may be ciliated.

Fibrous wall outside this.

Treatment:—

Remove tooth.

Open up cyst, *viâ* sulcus, by removal of outer wall.

Evacuate contents.
Thoroughly scrape away lining membrane.
Swab over with pure carbolic acid.
Pack tightly to arrest hæmorrhage.
Later pack more lightly with antiseptic dressing to promote growth of granulation tissue from the bottom, and thus by frequent dressing (daily), cavity will fill up.

d. **Exostosis** (*Productive Periodontitis*).

Outgrowth of cementum on root of tooth.

Causes:—

Chronic periodontitis—rather of the non-suppurative variety; especially that which is kept up by extension of inflammation through the apical foramen from a *chronic* pulpitis.

E.g. Large metal filling near pulp.
Erosion.
Attrition, &c.

Site:—

Any tooth, but especially molars.

Symptoms:—

Often none.
Slight pain on percussion.
Dull pain.
Neuralgia.

Treatment:—

Remove cause if possible.
Counter-irritants to gum.
Removal of tooth.

Microscopical Appearances:—

Lacunæ very variable in size and shape.
Canaliculi very variable in length and direction.
"Lines of Salter" very prominent.
Vascular canals occasionally present.
"Sharpey's fibres" sometimes well marked.

Varieties :—

a. Macroscopical—

1. Smooth.
2. Irregular.
3. General.
4. Nodular.
5. Bulbous (at apex).
6. "Candle grease."
7. Fusing two teeth together (false gemination).

b. Microscopical (according to predominance of one or other of its elements)—

1. Granular.
2. Lacunar.
3. Laminar.
4. Vascular.
5. Fibrillar.

Inostosis.

See below under "Absorption of Root."

Absorption of Root (*Rarefying Periodontitis*).

Strictly analogous to that of the Temporary Dentition. Large giant cells found fitting into Howship's Lacunæ. May be any extent, even opening pulp chamber. According to the way the character of the inflammation changes from time to time, so this process of absorption may alternate with the deposition of cementum. And if the absorption has extended sufficiently to attack the dentine, then cementum may be deposited later in the cavity thus formed in the dentine. This microscopically gives rise to an appearance named *Inostosis.*

Cementum may actually be deposited in the pulp chamber, if that has been opened up by the process.

GENERAL SUPPURATIVE CERVICAL PERIODONTITIS (*Pyorrhœa Alveolaris*)

Causes :—

Local—

1. Primary Gingivitis, extending to Periodontal Membrane.
2. Specific Germ (for Bacteriology, see above).
3. Tartar.

General—

4. Gout.
5. Other diatheses.
6. After fevers.

Predisposing—

7. Abnormal articulation.
8. Regulating teeth.

Site :—

Any teeth, but especially lower incisors.

May extend all round mouth.

May be limited to one aspect of teeth, or extend right round them.

Often seems to spread from one to another.

Symptoms :—

1. Pain and soreness of gums.
2. Congestion of gums at margins.
3. Pus discharging round neck of tooth.
4. Separation of gum from tooth, forming pockets.
5. Recession of gum and alveolar process.
6. Teeth loosening.
7. Ring of calculus, round neck of tooth, under margin of gum (deposit from pus).

Above symptoms vary in different patients and also from time to time in the same patient, according to both the virulence of the poison and the susceptibility of the patient at the moment. And often with long-standing cases the severity will vary

directly with the general health of the patient. It is most often found in rather a chronic condition.

Pathology:—

Absorption of alveolar margin.

Slight thickening of process just in advance of absorption.

Microscopically—

Hypertrophied papillæ in gum.

Dilated capillaries.

Enlarged epithelium.

Treatment:—

1. Remove any tooth that is hopelessly loose.
2. Scale thoroughly below gum margin.
3. Syringe out pockets with strong antiseptic (see Chapter xxiv.).
4. *Drugs,* directly applied to pockets—
   Tannic acid, if very sore.
   Copper sulphate (powdered), if very chronic.
   Protagal, 8 per cent.
   Trichloracetic acid—apply gently with wood point.
   Use either about twice a week.
5. Electrolysis with tincture of iodine.
6. Saturated solution of tannic acid in spirit, *rubbed* on the gums with the finger night and morning, is a useful adjunct to any of above treatments.
7. Good antiseptic and astringent mouth wash.
8. Vaccine treatment to improve the opsonic index in bad cases (Goadby).
9. Wire or splint teeth together to give periodontal membrane a rest.
10. Devitalise pulps of affected teeth in very chronic cases, in order to increase vascularity of periodontal membrane.

GENERAL PERIODONTITIS

Causes:—

1. Various diatheses—
   - Gout.
   - Rheumatism.
   - Syphilis.
   - Diabetes.
   - Malaria.
   - Scurvy.
2. After—
   - Exanthematous fevers.
   - Influenza.
3. Prolonged administration of mercury.
4. Fumes of phosphorus.
5. Extension from Pyorrhœa Alveolaris.
6. Injury—
   - Severe blow on jaw.
   - Partial dislocation of series of teeth.

Symptoms:—

Same as for Chronic Local Periodontitis, except that it occurs on several teeth at once.

Treatment:—

1. Remove and treat any cause.
2. Counter-irritants to gum.
3. Strong antiseptic and astringent mouth wash.
4. Purges.
5. Tonics.

CHAPTER VIII

INJURIES TO THE TEETH

1. Concussion.
2. Dislocation.
3. Fracture.
4. Dilaceration.
5. Erosion.
6. Attrition.
7. Abrasion.

1. Concussion

Causes :—

Direct blow.
Knocking teeth together.
Biting hard substance.

Results :—

Periodontitis—
a. Immediate.
b. Later, following death of pulp.

Pulpitis—
From the jarring.

Death of pulp by rupture of apical vessels. (This leads to discolouration of tooth.)

Treatment :—

Counter-irritants to gum.
Remove pulp, if pulpitis persistent or pulp dead.

2. Dislocation

Degrees :—

a. Partial.
b. Complete.

a. **Partial.**

Treatment :—

Replace tooth in correct position.
Fix with ligature or splint.
Counter-irritants to gum.

b. **Complete.**

Treatment :—

If during extraction—replace at once.
Otherwise remove pulp, fill canal, sterilise in mild antiseptic, and replace tooth and fix with splint.
Counter-irritants to gum.

3. Fracture

Causes :—

Blow.
Extraction.
Mastication.

Degrees :—

a. Not involving pulp.
b. Involving pulp.

a. **Not Involving Pulp.**

Symptoms :—

Exposed dentine sensitive.
Hyperæmia of pulp.
Periodontitis later.

Treatment :—

Alcohol or zinc chloride to dentine.

Strike up cap and cement on as temporary measure if sensitiveness persistent, as important to save pulp if root not complete.

Counter-irritants to gum.

If pulp suppurates, remove pulp if root complete ; otherwise remove tooth.

b. **Involving Pulp.**

Treatment :—

If age such that root not complete, remove tooth and let space close up, bearing in mind sex of patient and any evidence of irregularity.

If root complete, remove pulp, and prepare for crowning, if direction of fracture will allow of this. Otherwise extract tooth.

Fracture of tooth high up may unite sometimes, if kept quiet, by growth of secondary dentine and cementum.

4. Dilaceration

A distorted condition of tooth caused by injury to the same during development.

Characteristics :—

1. Common in front of mouth, being more liable to injury.
2. Crown or root or both affected, according to time and nature of injury.
3. Any degree of distortion may be found, from a mere bend to a much disordered tooth. There is often a bulging at the bend.
4. Sometimes marked mobility.
5. Generally erupt abnormally, and may not erupt at all.

5. Erosion

Characteristics :—

1. Saucer-shaped cavities, deepest near gum.
2. Generally labial aspect near gum.
3. Cutting edges sometimes involved.
4. Surface hard and polished.
5. One or more teeth affected.

Causes : —

1. Entirely abrasion from toothbrush and powders (Miller).
2. Acid secretion from buccal mucous glands, viz. acid sodium phosphate.
3. Potassium and ammonium sulphocyanide in saliva.
4. Associated with gout.

Microscopical Appearances :—

Abrupt termination of dentinal tubes, which are otherwise normal.

Zone of translucency under cavity.

No pigmentation.

Secondary dentine in pulp.

Treatment : –

Fill, if convenient, with gold or porcelain.

Silver nitrate or zinc chloride or milk of magnesia, if sensitive.

Softer toothbrush and light powder.

Cauterisation of buccal glands has been suggested.

6. Attrition

Wearing down of teeth upon one another.

Characteristics :—

Few or all teeth affected.

Any degree up to wearing down level with gum.

Causes :—

Edge-to-edge bite or other irregularity.
Harsh character of food.
Want of density of tooth substance.
Habit of grinding teeth together.
Gouty people are frequent subjects.

Pathology :—

Secondary dentine in pulp cavity.
Pulpitis sometimes.
Exposure of pulp rarely.

7. Abrasion

Wearing of teeth by friction with foreign body.

Causes :—

Denture.
Pipe, especially clay pipe.
Toothbrush (see *Erosion*).
Occupations, where things continually held between the teeth.

CHAPTER IX

DISEASES OF GUMS AND MUCOUS MEMBRANE OF MOUTH

A. Gingivitis.
B. Stomatitis.
C. Tumours.
D. Hypertrophy of Gums.
E. Scurvy of Gums.
F. Addison's Disease.
G. Purpura.
H. Pemphigus.
I. Lead Poisoning.

A. GINGIVITIS (*Inflammation of Gums*)

Causes :—

Constitutional—

Gout.
Rheumatism.
Syphilis.
Diabetes.
Malaria.
Indigestion.
Alcoholism.

Medicinal—

Mercury.
Potassium iodide.

Septic—

Tartar.
Roots of teeth.
Want of cleansing.
Dentures.

Traumatic—

Toothbrush.
Too rapid regulating.

Varieties :—

1. Confined to gum margins.
2. General.

Symptoms :—

Gums red, swollen, and soft, especially margins.
May be hypertrophied between teeth.
Often dark-coloured calculus on teeth under free margin of gum, deposited from pus, which oozes up.
Bleed easily.

Treatment :—

Treat cause.
Pack powdered copper sulphate under gum twice a week.
Rub on tannic acid in spirit twice a day.
Antiseptic and astringent mouth-wash.

Results :—

Cervical periodontitis, if untreated.
Recession of gum, if recurring.

B. STOMATITIS

(*Inflammation of Mucous Membrane of Mouth*)

Varieties :—

1. Simple.
2. Mercurial.
3. Follicular.

4. Ulcerative.
5. Gangrenous.
6. Parasitic.
7. Syphilitic.

1. Simple Stomatitis

Causes :—

Uncleanliness.
Tobacco smoke.
Gastric and intestinal troubles.
Fevers.
Associated with nasal catarrh.

Symptoms :—

Mucous membrane tender and bleeds easily.
At first dry, and then wet with sticky secretion which later becomes purulent.
Congested margin to gums.
Gums and sulcus pale and mottled—the mottling being due to sodden epithelium being rubbed off the tips of enlarged papillæ, leaving red patches.
Fœtid breath.
General malaise.

Treatment :—

Antiseptic and astringent mouth-wash.
Purge.
Tonic—Strychnine and Iron.

2. Mercurial Stomatitis

Cause :—

Mercury given medicinally to excess.

Symptoms :—

All symptoms as for *Simple Stomatitis.*

Also—

Teeth and gums tender.
Pain on eating.
Metallic taste.
Salivation.
Periodontitis.
Ulceration of margin of gums.
Sloughing and necrosis.

Treatment :—

Stop course of mercury.
Antiseptic and astringent mouth-wash.
Application of tincture of iodine.
Potassium chlorate and potassium iodide internally.
Purge.

3. Follicular Stomatitis

Symptoms :—

Inflammation of mucous follicle, turning to small ulcer with sharply cut edges and zone of redness.
Very painful.
May be single or multiple.
Most often occur in sulcus.

Treatment :—

Touch with copper sulphate or silver nitrate.

4. Ulcerative Stomatitis

Causes :—

Young children out of health.
Uncleanliness.

Symptoms :—

Starts in front of mouth and extends as line of ulceration along margin of gum, and later on along cheek from contact, leaving ulcer with sharp, irregular edge and bluish margin.
Unilateral as a rule.
Little pain at first.

Salivation.
Fœtid breath.
Swelling of cervical lymphatic glands.
Loosening of teeth.
Sloughing and necrosis.

Treatment :—

Antiseptic and astringent mouth-wash.
Swab out with—
Pot. Chlor. ℥i.
Ac. Hydrochlor. dil. ℥ss.
Aq. ad. ℥xx.
Apply—
Tannic acid. } āā. three times a day.
Dried alum. }
Touch with silver nitrate twice a week.
Potassium chlorate internally in syrup.
(gr. i. for every year of child's age up to gr. x.)
Purge.
Tonic.

Results :—

Loss of teeth.
Necrosis.
Lymphatic abscess.
Septicæmia.
Pyæmia.
Septic gastritis.
Septic pneumonia.
Inanition.
Fibrous bands in cheek, on healing.

5. Gangrenous Stomatitis
(*Cancrum Oris*) (*Noma*)

Causation :—

Generally in children (2–6 years).
After fevers.
Idiopathic.

Pathology :—

Due to a *diffuse capillary thrombosis*, induced by germs.

Symptoms :—

Great swelling of face, but very little œdema.
Ulcer on mucous membrane inside cheek.
Dark spot outside at same place.
Rapid destruction of cheek, making opening, which may be large, and even whole cheek may be destroyed.
Drowsiness and exhaustion and possibly death.
May undergo temporary arrest.

Treatment :—

After drying, apply one of following—
a. Carbolic acid.
b. Nitric acid.
c. Actual cautery.
Excise ulcer and perform plastic operation.
Inject pure carbolic acid around ulcer.
Prevent septic matter from entering air passages, &c., by giving good mouth-washes, and applying antiseptic dressing.
Support general system—
Nourishing food.
Stimulants.
Tonics.

Results :—

Same as for *Ulcerative Stomatitis*.

6. Parasitic Stomatitis—Thrush

Causation :—

Occurs in babies mostly.
Occurs in old people, usually just before death, in wasting diseases.
Due to *Oidium albicans* = mycelium of fungus.

Symptoms :—

Resembles *Simple Stomatitis* when it begins, but patches rapidly cover with exudation, forming small white patches.

These may peel after a few days, but cover over again.

Treatment :—

Wipe mouth round after every meal.
Borax gently rubbed on.
Alum gently rubbed on.
Sulphurous acid applied.

7. Syphilitic Stomatitis

a. Primary Ulcer—

May occur anywhere in mouth.

b. Secondary Lesions—

Mucous tubercles, especially on edges of tongue, and angles of mouth, and inside cheeks.

May be shallow ulcers.

c. Tertiary Lesions—

Swelling first (*gumma*), which breaks down, leaving large, deep ulcer.

C. TUMOURS OF GUMS AND MUCOUS MEMBRANE OF MOUTH

Forms :—

1. Polypus.
2. Epulis.
3. Papilloma.
4. Nævus.
5. Epithelioma.
6. Sarcoma.
7. Gumma.

1. Polypus of Gum (*Local Hypertrophy*)

Causes :—

Tartar.
Ragged edge of tooth.
Clasp of denture, &c.

Symptoms :—

Generally between two teeth.
May be pedunculated or not.
Very tender.
Bleeds easily.

Diagnosis :—

From polypus of pulp.
From epulis.

Treatment :—

Cut off.
Burn out with red-hot burnisher or cautery.
Cauterise away with ethylate of sodium, &c.
Push out with G. P. if small.

2. Epulis

Symptoms :—

Generally between two teeth.
Pedunculated and lobulated.
Springs from junction of periodontal membrane with periosteum.
Usually slow growing, but may be rapid.
May attain large size.
May ulcerate.
More common in women.

Pathology :—

3 *varieties*—

a. Fibrous tissue with small round cells.
b. Fibrous and elastic tissue.
c. Fibro-sarcoma with myeloid cells.

Any of above may contain besides small pieces of bone or root, which have become detached by absorption.

Treatment :—

Cut off and scrape base freely and cauterise with pure carbolic acid.

Remove tooth from which it springs, if growth large.

3. Papilloma

Pathology :—

Hypertrophy of papillæ of mucous membrane.

Analogous to a *Wart*.

Treatment :—

Remove and cauterise.

4. Nævus

Symptoms :—

Flat, red, and only slightly raised.

Begins as red pimple.

Seldom larger than half an inch across, but may be large.

Bleeds easily.

Blood can be pressed out, but returns when pressure relaxed.

Pathology :—

Hypertrophy and multiplication of capillaries.

Treatment :—

Remove and cauterise.

5. Epithelioma 6. Sarcoma 7. Gumma

See General Surgery.

D. HYPERTROPHY OF GUMS

Cause :—

Congenital, starting in quite early life.

Symptoms :—

Masses of gum tissue, growing up round teeth, and even covering them, and sometimes protruding from mouth.
Masses may be lobulated or have smooth appearance.
Darker than normal gum.
Very vascular and bleed easily.
Often discharge.
Pus oozing up round teeth.
Fœtid breath.

Treatment :—

Cut off, and remove surface of alveolar process as well.
After removal, cauterise with actual cautery, because of extensive bleeding.

Pathology :—

Firm fibrous stroma, springing from alveolar process.
Much glandular tissue.
Large vascular papillæ.
Epithelium often very thick.
Alveolar process generally enlarged too.

Transparent Hypertrophy

Symptoms :—

Pale blue cord, running along gum margin.
Teeth loosen and fall out.
Cuts like cartilage, and does not bleed.
Very rare condition.

E. SCURVY OF GUMS

Symptoms :—

Gums over process—*red* and swollen.
Rest of mucous membrane and lips—*white*.
Bleeds easily.
Teeth loosen.
Ulceration of gums.
Sloughing and necrosis.

F. ADDISON'S DISEASE

Pigmented patches found in gums.
Associated with pigmentation elsewhere.

G. PURPURA

Hæmorrhages under mucous membrane of mouth.
Associated with Purpura elsewhere.

H. PEMPHIGUS

Blebs on mucous membrane of mouth, especially palate and cheeks.
Associated with Pemphigus elsewhere.

I. LEAD POISONING

Symptoms :—

Blue line upon gums, generally round lower incisors.
Absent if teeth are absent.
Discoloured patches on mucous membrane of cheeks.
Colic.
Wrist drop.
Constipation.
Metallic taste.
Fœtid breath.
General debility and anæmia.

Diagnosis :—

Blue line has to be diagnosed from—

a. Deposit of white lead, not in but on gums.
b. Charcoal tooth-powder.
c. Carbon dust in miners.
d. Copper and bismuth poisoning (rare).
e. Black tartar under margin of gums.

Sources of Lead Poisoning :—

1. Lead and its salts in factories.
2. Occupations, &c.
   a. Painters.
   b. Potmen.
   c. Compositors.
   d. Brussels lace makers.
   e. Glazed card makers.
3. Applications—
   a. Ointments.
   b. Hair dyes.
4. Water—
   Leaden cisterns and pipes.
5. Food.

CHAPTER X

SALIVARY CALCULUS

Salivary Calculus is a deposit from the saliva of salts, held in solution by excess of carbon dioxide gas, which is liberated on exposure to the air and thus the salts are deposited.

Sites :—

1. In ducts and glands.
2. On teeth. } Tartar.
3. On dentures. }

1. In Ducts and Glands

Results :—

a. Retention cyst.
b. Suppuration of cyst.
c. Salivary fistula.
d. Ulceration of duct.

2. On Teeth

Sites :—

Especially—
a. Lingual aspect of lower incisors.
b. Buccal aspect of upper molars.

Being opposite salivary ducts.

But may occur anywhere in mouth, and is occasionally sufficient to completely hide the teeth of one side.

Conditions favouring Accumulation of Tartar:—

a. Lack of Mastication.

Painful tooth—

(i) Exposed pulp.
(ii) Periodontitis.
(iii) Polypus of pulp or gum.

Sore gums.
Loss of antagonising teeth.
Habit of using one side only.

With any of above, tartar *may* collect over all the teeth on one side.

b. Lack of brushing.

c. Rough surfaces—

(i) Cavities.
(ii) Pits.
(iii) Bad fillings.
(iv) Dentures.

d. Irregularities in position.

e. Condition of saliva (varies in individuals).

f. Condition of health. (?)

g. Gout. (?)

Effects:—

Gingivitis.
Recession of gum.
Periodontitis.
Absorption of alveolus.
Looseness of teeth.
Pyorrhœa.
Sore tongue.
Lodgment of food.
Foul breath.
Gastric catarrh.

Composition :—

a. Salts.

Carbonates of Calcium and Magnesium—
Chief salts from parotid, *e.g.* on molars.

Phosphates of Calcium and Magnesium—
Chief salts from sublingual, *e.g.* on lower incisors.

Chlorides of Sodium and Potassium—
Small quantities from both glands.

Sulphocyanide of Potassium—
Traces only.

b. Animal Matter.

Epithelium.
Food.
Pus cells.

c. Other Matter.

Water.
Mucus.
Ptyalin.
Pigment (smoke, &c.).

Quantitative Analysis shows great variation.

Roughly—

Salts .	.	80 per cent.
Water and Mucus	12	,,
Animal Matter	7	,,
Ptyalin .	.	1 ,,

Hard tartar will contain rather *more* salts.

Soft tartar will contain rather *less* salts.

Treatment :—

Removal by "Scaling."

Principles of Scaling :—

a. Remove every particle and polish.

b. Chip off in as large pieces as possible, leaving less to scrape.

c. Push towards gum when large amount.

d. Pull away from gum when small amount.

Precautions in Scaling :—

a. Instruments to be sterile and sharp.
b. Avoid injuring gum.
c. Avoid injuring enamel.
d. Support loose teeth.
e. Prevent patient swallowing particles.
f. Give mouth-wash afterwards.

CHAPTER XI

SALIVARY FISTULA

Varieties:—

1. Connecting mouth with exterior.
2. Connecting salivary duct with exterior.

1. Connecting Mouth with Exterior.

Causes:—

a. Alveolar abscess bursting externally and internally.
b. Necrosis.
c. Bad fracture.
d. Slipping of elevator.
e. Cancrum oris.
f. Syphilitic ulceration.

Any of which may be means of saliva finding its way through.

Treatment:—

a. Treat cause.
b. Plastic operation.
c. Pass seton through (old treatment).

2. Connecting Salivary Duct with Exterior.

Causes:—

Blocking of duct by—

a. Calculus.
b. Cicatrix.
c. Growth.

Opening of duct by—

a. Knife.

b. Suppuration.

c. Ulceration of malignant growth.

Treatment :—

Remove calculus or growth.

Cause duct to open inside.

Plastic operation outside.

CHAPTER XII

ODONTOMES

An odontome is a neoplasm composed of dental tissues in varying proportions and different degrees of development, arising from a tooth germ or a tooth still in its process of growth (Bland Sutton).

Classification (Bland Sutton):—

1. Aberrations of *enamel organ*—
   Epithelial odontome.

2. Aberrations of *follicle*—
   a. Dentigerous or follicular odontome.
   b. Fibrous odontome.
   c. Cementoma.
   d. Compound follicular odontone.

3. Aberrations of *papilla*—
   Radicular odontomes.

4. Aberrations of *whole tooth germ*—
   Composite odontomes.

N.B.—The above is the accepted classification, but it will be seen that the different forms of odontomes merge so much into one another that it makes a comprehensive classification a very difficult matter.

1. ABERRATIONS OF ENAMEL ORGAN

Epithelial Odontome

Site :—

Generally lower molar region.

Pathology :—

On section—numerous cysts of various sizes, separated by fibrous or bony septa.

Contain brown mucoid fluid.

Walls and septa, on microscopical examination, show branching columns of epithelium with fibrous tissue stroma.

2. ABERRATIONS OF DENTAL FOLLICLE

a. Dentigerous Cyst or Follicular Odontome

Site :—

Lower molar region.

Antral region.

Pathology :—

Excessive secretion of fluid between the developing tooth and the follicle wall, gradually enlarging its crypt and body of jaw.

Fluid is yellowish in colour, and may contain cholesterin.

Walls are fibrous and usually have *epithelial lining*.

Generally contains tooth, either loose or partially fixed in floor, or may be inverted.

May suppurate.

N.B.—The epithelial lining may possibly indicate that the enamel organ may have aberrated after forming the enamel.

b. Fibrous Odontome

Pathology :–

Hypertrophy of tooth sac.
May be laminated on section.
May have tooth in centre.

c. Cementoma

Pathology :—

Calcification *en masse* of a fibrous odontome.
Formed of cementum, since follicle forms that tissue.

d. Compound Follicular Odontome

Pathology :—

Calcification of a fibrous odontome in a sporadic manner, with sometimes cystic formation.
Made up of masses of cementum and bone, embedded in loose fibrous tissue, or lying loose in fluid, when the follicle has also become cystic.
N.B.—Certain cystic odontomes, containing many denticles of enamel, dentine, and cementum, although sometimes classified under this heading, because they are cystic, would perhaps be better classified as "Composite Odontomes."

3. ABERRATIONS OF DENTINE PAPILLA

Site :—

Lower jaw.

Pathology :—

Due to an outgrowth of the dentine papilla, after the completion of the crown and during or before the formation of the root, and then partial or complete calcification.

Thus, according to the predominance of the tissue formed, there may be—

a. Radicular dentoma.
b. Radicular osteo-dentoma.
c. Radicular cementoma.

The cementum in the last may be formed by the diseased dentine papilla, or by the surrounding follicle, enlarged by the dilatation of the papilla

N.B.—Many irregularly-shaped teeth with malformed crowns may be classified as this form of odontome, having possibly arisen by an outgrowth of the dentine papilla before the commencement of calcification of the crown.

4. ABERRATIONS OF WHOLE TOOTH GERM

Composite Odontome

Pathology :—

Due to irregular growths of all parts of the tooth germ, forming irregular masses of calcified tissue.

On section they contain enamel, dentine, and cementum, all arranged quite indefinitely, either in one solid mass, or a large number of smaller masses or denticles.

Symptoms of all Odontomes :—

1. Often no pain.
2. May be merely sense of uneasiness or neuralgic pain.
3. *Tumour*, generally encroaching upon alveolus, increasing very slowly, and varying in physical characters, according to variety of odontome.

4. May become inflamed, and finally suppurate and point, leaving sinuses.
5. Generally tooth missing, but not always.
6. Although starting to grow at usual time of development of tooth, not as a rule noticed till about 15 to 20 years of age.

CHAPTER XIII

ODONTALGIA AND NEURALGIA

ODONTALGIA

Odontalgia is pain in or around a tooth.

Forms :—

1. Local.
2. Referred.

1. Local

Causes :—

Morbid condition of *that particular tooth.* (Fully considered in foregoing pages.)

2. Referred

Causes :—

Exactly the same as for neuralgia. (See below.)

N.B.—If the cause is *peripheral*, it is always on the same side as the pain, which is never referred across the middle line.

NEURALGIA OF THE 5TH NERVE

Neuralgia is pain in the course of, or in the area of distribution of, one or more of the branches of the nerve.

Causes :—

Predisposing :—

a. Overwork.
b. Neurotic temperament.
c. Reduced vitality.

Exciting :—

a. Peripheral—

(i) Affections of pulp.
(ii) „ periodontal membrane.
(iii) „ periosteum.
(iv) „ gum.
(v) „ mucous membrane of mouth, nose, or antrum.
(vi) „ skin.
(vii) „ eye.
(viii) „ viscera, uterus, &c.

b. Course of Nerve—

(i) Injury.
(ii) Pressure—
Wisdom tooth.
Tumours.

c. Cerebral—

(i) Degenerations.
(ii) Tumours.
(iii) Hysteria.

d. Systemic—

(i) Gout.
(ii) Rheumatism.
(iii) Syphilis.
(iv) Malaria

e. Idiopathic.

Dental Causes :—

1. Caries.
2. Metal fillings too close to pulp.
3. Oxychloride fillings.
4. Erosion.
5. Attrition.
6. Abrasion.
7. Fracture of tooth.
8. Exposure of cementum.
9. Calcification of pulp.
10. Degeneration of pulp.
11. Periodontitis from any cause, especially the chronic form.
12. Exostosis.
13. Buried root.
14. After extraction—
    Tearing of nerve.
    Healing of tissues.
15. Erupting teeth.
16. Impacted wisdom teeth.
17. Absorption of 2nd molar by 3rd molar.
18. Inflammation of gums.
19. Growths, odontomes, and cysts.

Diagnosis *re* Teeth :—

Probe all round and under gums.
Suspect any filling.
Test each tooth with heat and cold.
Tap all teeth.
Press teeth in all directions.
Suspect pulp of lately dying.
Note wisdom teeth—whether erupted.
Note position of pain—
    Upper teeth—infra- and supra-orbital.
    Lower teeth—ear, neck, and shoulder.
    Upper and lower teeth—parietal region.
Failing the teeth, look for other cause.

CHAPTER XIV

SECONDARY AFFECTIONS DUE TO DISEASED TEETH

1. **Direct Septic Infection**—*i.e.* extension of inflammation.
   - Empyema of antrum.
   - Necrosis.
   - Post-pharyngeal abscess.
   - Œdema glottidis.
   - Cellulitis of neck.

2. **Remote Septic Infection.**
   - Lymphadenitis. } *Viâ* lymphatics.
   - Lymphatic abscess. }
   - Meningitis. } *Viâ* facial vein.
   - Thrombosis of sinuses. }
   - Septic gastritis. } By swallowing septic matter.
   - Appendicitis. }
   - Septic pneumonia. By inhaling septic matter.

3. **Septic Absorption**—by toxins or micro-organisms getting into blood stream.
   - Sapræmia.
   - Septicæmia.
   - Pyæmia.
   - Septic Anæmia.
   - Septic Neuritis.
   - Puerperal fever.
   - Eruptions of skin.

4. **Nervous Irritation**—by reflex irritation of sensory, motor, trophic or vaso-motor nerves.
   - Neuralgia.
   - Epilepsy.
   - Hysteria.
   - Trismus.
   - Spasm of facial muscles.
   - Wry neck.
   - Paralysis.
   - Herpes.
   - Various diseases of eye.
   - Various diseases of ear.

5. **Tissue Irritation**—by continuous irritation from rough surface, combined with sepsis.
   - Ulcer of tongue.
   - Epithelioma.

6. **Malnutrition**—from want of mastication.
   - Indigestion.
   - Wasting.

CHAPTER XV

EMPYEMA OF ANTRUM

Causes :—

1. Extension of inflammation from roots of teeth—any upper tooth, but especially 1st molar.
2. Extension of inflammation from nasal cavity.
3. Extension of inflammation from— } *Viâ* nose.
   Frontal sinus.
   Ethmoidal sinus.
4. Alveolar abscess bursting into antrum, instead of in sulcus.
5. Passage of foreign body—
   a. Root.
   b. Food.
   c. Toothpick.
6. Necrosed bone ; syphilitic, &c.
7. Blow on face.
8. Operations on face.
9. Fracture of maxilla.
10. Cold. (?)

Symptoms :—

Acute condition—when ostium maxillaire will probably be closed by swelling of mucous membrane—

Pain over forehead and face.

Infra-orbital neuralgia.

Throbbing—worse when recumbent.

Tender to pressure.

Bulging of antrum, especially on face.
Parchment crackling if bone thinned.
Fluctuation if bone absorbed away.
Pyrexia.
Rigor.
Malaise.

Chronic condition—when ostium maxillaire will probably be open—

Pain as above, though less.
Discharge from one nostril if ostium not too high up.
Surging of fluid, heard by patient only.
Smell, noticed by patient only.
Indigestion from swallowing pus.
Pus coughed up in the morning from pharynx.

Diagnosis :—

Means :—

1. Look for cause.
2. Position of patient—head forward and affected side uppermost, pus may trickle from nostril.
3. Speculum in nose—pus may be seen in middle meatus.
4. Catheterisation of ostium maxillaire by nose.
5. Transillumination by electric light in mouth or pharynx. Infra-orbital zone of light if antrum normal.
6. Aspiration with needle and syringe—through inferior meatus.

Treatment :—

1. *Open antrum* and drain—
   a. Socket of extracted tooth. Buccal root of 1st molar for preference.
   b. Canine fossa.
   c. Under malar bone.
   d. Inferior meatus.

Open by means of—

Ordinary gimlet.
Antral gimlet with guard.
Burr or drill on engine.
Straight elevator.

Opening to be made *large* (as little finger), if special plug or tube is to be made.

Opening to be made *same size as tube*, if an already made tube is to be used.

2. *Syringe* forcibly at once with any mild antiseptic solution by means of antral syringe.

Get the solution and pus to pass out through nose if possible.

3. *Drain*—

a. By putting in at once a temporary drain,

e.g. piece of catheter,
piece of rubber tubing,
piece of gauze,

and proceed to make tube or plug to pass through hole into antrum, and retain by a flange if in sulcus, or a denture if in alveolus.

b. By putting in at once a—

Symond's tube,
Ackland's tube,
McGavin's tube,

and leave in, syringing by means of the respective special syringes.

Whichever means of drainage is used the antrum must be syringed once or twice a day with any mild antiseptic forcibly, so as to penetrate into all the recesses. Change of antiseptic is often beneficial.

In favourable cases the above should effect a cure in 3 or 4 weeks. But the more chronic cases may go on discharging for months, years, or even be absolutely incurable by this means. In any case the hole should be kept open, as long as there is any discharge.

4. *Further treatment* for chronic cases.

Open more widely and pack with lint dipped in carbolic acid (1 in 20) to destroy diseased mucous membrane and promote growth of fresh one, or swab over with pure carbolic acid.

Pack daily.

May be opened more widely still, and scraped and tightly packed. In this case antrum fills up with granulation tissue and probably becomes obliterated.

This latter method of treatment is the one that is gaining more favour every day with modern surgeons as the *primary* measure in treating antral disease.

CHAPTER XVI

NECROSIS OF THE JAWS

Sites :—

Either jaw, but more frequently mandible, because—

a. More exposed to injury.

b. More compact bone.

c. Less blood supply.

Causes :—

1. Extension of inflammation from alveolar abscess—
   By vessels of bone becoming inflamed and thrombosed.
2. Escape of arsenic dressing through apex of tooth or on to gum—
   By corrosive action of drug.
3. Blow on jaw—
   By periosteal abscess stripping off periosteum.
4. Fracture—
   By—
   a. Directly cutting off blood supply.
   b. Becoming septic from mouth.
5. Inhalation of fumes of yellow phosphorus—
   By direct corrosive action.
6. Administration of mercury—
   By acting through the system.
7. Exanthematous fevers—
   By periostitis probably during acute stage.

8. Ulcerative stomatitis and cancrum oris—
   By suppuration of periosteum.
9. Syphilis—
   By breaking down gummatous infiltration.
10. Tubercle—
   By carious disintegration.
11. Malignant disease—
   By extension into bone.
12. Actinomycosis—
   By extension into bone.
13. Idiopathic.

Symptoms :—

Gums swollen and tender.
Abscess pointing.
Sinuses in mouth and on face.
N.B.—Multiple sinuses on face almost invariably indicate necrosis.
Teeth loose.
Breath fœtid.
Skin red and œdematous.
May end fatally.

Treatment :—

Remove cause.
Hot fomentations in mouth.
Antiseptic mouth-wash.
If abscess shows signs of pointing on face, apply hot fomentations, or open externally.
If possible, get free exit for pus in mouth.
Dress external sinuses.
Remove sequestrum when loose and covered only by soft tissues (ascertained by probing sinuses, two at once if possible, with stiff probes, to feel if bone rocks).
Give periodical purges.

Phosphorous Necrosis

Cause :—

Fumes of yellow phosphorus enter deep parts by—

a. Breach of periosteum.

b. Socket of recently extracted tooth.

c. Exposed pulp.

Site :—

Body of jaws—

Upper and lower.

Symptoms :—

Great pain, beginning often with toothache.

Great swelling.

Great suppuration and several sinuses.

Great constitutional disturbance.

Pathology :—

Sequestrum has upon it a "pumice stone deposit," formed by periosteum.

Sequestrum also generally presents large interlacing Haversian canals running at right angles to direction of bone.

Extensive production of new bone.

Exanthematous Necrosis

Causes :—

Any of the exanthematous fevers, but especially—

Scarlet fever.

Measles.

Generally occurs about 4th year and never after 10th year.

Site :—

Alveolar portion of jaw.

Usually symmetrical if near front of mouth.

Symptoms :—

Breath very fœtid—often the only symptom, till gums observed.
Insidious onset.
No pain as a rule.
No swelling.
Pus oozing up round teeth, which loosen.
Gums strip off alveolar process in a vertical direction.

Pathology :—

Sequestrum is a dirty yellowish colour, and may be dark brown if exposed to air.
Margins ragged.
Worm-eaten appearance.

CHAPTER XVII

FRACTURES OF THE JAWS

Causes :—

Direct Violence—

Blows.
Kicks.
Falls.
Gunshots.
Violent extraction.

Indirect Violence—

Coughing, &c., when jaw weakened by disease.

Sites :—

Mandible—

Anywhere, but especially—
Canine region.
Symphysis.
Angle.
Molar region.
Neck.

Maxilla—

Generally alveolar portion, and especially in front.

Symptoms :—

Pain in region of fracture and worse on movement.
Loss of power.

Deformity—line of teeth altered, due to displaced fragment.
Swelling.
Crepitus.
Mobility of fragments.

Causes of Displacement :—

Voluntary muscular action—
Eating, talking, &c.
Involuntary muscular action—tonic spasm.
Swelling.
Obliquity of line of fracture.
Soft tissue between fragments.
Tooth between fragments.
Piece of bone between fragments.
Callus between fragments.

Treatment :—

a. Mandible

1. *Bandages.*

4-tailed.
Hamilton.
Either alone or in conjunction with chin boot of some sort.

To be used—
When fracture single, simple, and no displacement, and only very slightly movable.

2. *Hammond's* wire splint or modification, surrounding all teeth and wired to teeth with binding wire.

To be used—
When fracture single and anywhere *in front of* or in premolar region.
When little displacement and easily rectified.
When most of teeth are standing and firm.

3. *Tomes'* Splint.—Metal cap covering all the teeth, and pressed home with cement or G. P.

To be used—
When conditions are similar to those for a "Hammond," except that it can be used when fewer teeth are standing, and if they happen to be loose.

4. *Hayward's* Splint.—Vulcanite cap over all the teeth with wire wings projecting out of mouth along cheek. Pressed home with G. P., and bandaged under chin and over wire wings.

To be used—
As for Tomes' splint.

5. *Gunning's* Splint or modification.—Caps of vulcanite or metal covering both upper and lower jaws, fixed together at sides. Bandage also applied.

6. *Angle's* studded bands and wires.—Opposing teeth in upper and lower on each side are banded and wired together over the studs on the bands.

To be used—
When angle or vertical ramus fractured.
When more than one fracture.
When great difficulty in rectifying displacement.
With these forms—
Upper jaw forms a fixed point for the lower to be held motionless against it.

7. *Surgical wiring* of fragments.

To be performed—
When fragments displaced considerably.
When difficulty of retaining splints from any cause.

8. *General directions* to patient.

Jaw not to be moved, if bandaged up.

Soft food with Hammond's, Tomes', and Hayward's splints.

Slop diet with Gunning's splint and Angle's methods.

Good mouth-wash and toothbrush frequently used.

b. Maxilla

If slight—

Ligature teeth together.

If extensive—

Small Hammond's or Tomes' splint.

Complications of Fracture of Mandible.

Wounds of face.
Dislocation or fracture of teeth.
Abscess, necrosis, and sinuses.
Salivary fistula.
Injury to base of skull.
Injury to brain, through glenoid cavity.
Dislocation of jaw.
Irregular union.
Ununited fracture.
Splintering of bone.
Nerve injured or involved in callus—
    Anæsthesia.
    Neuralgia.
    Paralysis.
Infiltration of blood about base of tongue—
    Dyspnœa.
Hæmorrhage.
General—
    Shock.
    Septicæmia, &c.
    Erysipelas.
    Fat embolism.

CHAPTER XVIII

DISLOCATION OF TEMPORO-MAXILLARY ARTICULATION

Causes :—

Direct Violence—

Blows.
Kicks.
Falls.
Violent extraction.

Muscular Action—

Yawning.
Shouting.
Opening mouth wide to put large things in.

Predisposed to by—

Previous dislocation.
Weakly young subjects—loose joint.
Arthritis.

Varieties :—

Unilateral.
Bilateral.
Complicated with fracture.

Symptoms :—

Deformity of jaw.
Projection below malar bone—coronoid process.

Projection above zygoma — contracted temporal muscle.
Masseter muscle hard and prominent.
Hollow behind condyle.
Pain and swelling.
Dribbling of saliva.
Loss of power—speech and deglutition altered.

Treatment :—

Reduction at once by disengaging condyle from eminentia articularis, and application of 4-tailed bandage for some days.

CHAPTER XIX

INABILITY TO OPEN THE MOUTH
("Forced Closure")

May be—

1. Complete.
2. Incomplete.

Causes :—

1. Organic lesions.
2. Spasmodic conditions.
2. Traumatic conditions.

1. Organic Lesions :—

a. Infiltration of soft tissue around vertical ramus and angle with inflammatory products—

(i) Lower 3rd molar trouble.

(ii) Inflammation of lymphatic glands.

(Often incorrectly called Trismus.)

b. Diseases of joint—

(i) Acute or chronic arthritis.

(ii) Anchylosis—bony or fibrous.

c. Fibrous bands in cheek, *i.e.* cicatrices from previous ulceration, &c.—

Burns.

Lupus.

Caustics.

Cancrum oris.

Ulcerative stomatitis, &c.

d. Parotid and other tumours.

e. Mumps.

f. Deep-seated growths.

g. Exostosis of zygoma.

h. "Bony bar"—ossification of pterygo-maxillary ligament.

2. Spasmodic Conditions :—

a. Trismus—spasm of masticatory muscles.

Causes :—

Inflammatory conditions.
Erupting wisdom tooth.
Carious tooth.
Hysteria.

b. Tetanus—due to tetanus bacillus.

Closure of jaw may be first symptom, followed by spasm of other muscles.

c. Strychnine poisoning.

d. Hydrophobia.

3. Traumatic Conditions :—

a. Fracture—especially if severe.

b. Dislocation—jaw fixed open.

CHAPTER XX

SWELLINGS ABOUT THE JAWS

Diagnosis :—

First Determine—

1. Connected with soft parts only — freely movable over jaw.
2. Connected with jaw—
   - *a.* Attacked externally — circumscribed swelling.
   - *b.* Involving substance—fusiform swelling.

If involving substance of jaw, it is possibly of dental origin, therefore—

Next Determine—

1. Fluid swelling.
2. Solid swelling—
   - *a.* Innocent.
   - *b.* Malignant.

Fluid Swellings.

Chaarcteristics of All :—

1. Regular, smooth, and globular.
2. May fluctuate.
3. May be egg-shell crackling.

4. Thinner wall of jaw (outer plate) enlarging first.
5. Alveolar border not interfered with.

Besides the above points common to all fluid swellings in jaw, note also the following individual characters:—

a. Abscess.

Presence of root or dead tooth.
Presence of inflammation somewhere.
Short history (comparatively).
Varies in size, perhaps, or—
Came quickly and remained so.

b. Dental Cyst.

Presence of root or dead tooth.
No inflammation around (unless it suppurates later).
Slow, steady growth.

c. Dentigerous Cyst.

Absence of tooth from arch usually.
No inflammation around.
Very slow growth.
Age—15 to 20 years.
Skiagraph may show tooth.

d. Multilocular Cyst (Epithelial Odontome).

Absence of tooth from arch usually.
No inflammation around.
Very slow growth.
Age—15 to 20 years.
Tendency to be nodular.

e. Cyst of Independent Origin.

Mucous cyst of antrum.
No inflammation around.

Solid Innocent Swellings

Characteristics of All :—

1. Slow growth.
2. Solid feel—according to *kind* and *consistency*.
3. Alveolar border displaced little.
4. Affects one wall of jaw as much as another.
5. Little pain.

Forms :—

Fibroma.
Enchondroma.
Osteoma.
Myxoma.
Solid odontomes—
    Fibrous.
    Cementoma.
    Composite.
    Radicular.

Solid Malignant Swellings

Characteristics :—

1. Quick growth.
2. Solid or pulpy feel.
3. Alveolar border displaced irregularly.
4. Affects one wall more than another, but not necessarily the thinner.
5. Severe pain.
6. Early implication of skin.
7. Emaciation.
8. Œdema of face.
9. Enlarged glands—carcinoma.
10. Attacks of hæmorrhage if superficial.

Forms :—

Sarcoma.

Age—young or adults.
Tendency to bleed.

Carcinoma.

Age—adults or aged.
Glands affected.

CHAPTER XXI

CLEFT PALATE

Causes :—

1. Congenital.
2. Acquired.

1. CONGENITAL

Due to—

Arrest of development.

N.B.—Not merely an arrest of union, but also arrest of formation of tissue.

Varieties :—

Any extent of cleft, from a bifid uvula up to a complete cleft passing through alveolus on one or both sides. May then be associated with single or double hare-lip.

N.B.—With this latter the premaxilla is deformed and very much protruded.

Relation of teeth to cleft through alveolus :—

a. { Central on mesial side (lateral missing). / Canine on distal side. } Most common.

b. { Central on mesial side. / Small tooth — generally called precanine (? lateral) with canine on distal side.

c. { Lateral on mesial side. / Canine on distal side.

Effects :—

Deglutition interfered with little.
Speech interfered with great deal—
D pronounced as N.
B pronounced as M.
G and K are impossible.

Treatment :—

A. Operative.
B. Mechanical.

A. Operative—

Staphylorraphy—union of soft palate.
Uranoplasty—union of hard palate.

Very many different plastic operations of the above have been devised. Tendency now with most surgeons is to operate early, *i.e.* when patient is about 1 to 3 months old.

B. Mechanical—

a. Velum—

A soft, elastic, and movable appliance, fixed to the back of a denture, and which should imitate the natural soft palate.

Forms—

(i) *Sercombe's.*

Flap of sheet rubber cut out to cover cleft and extend to within quarter of inch of posterior wall of pharynx, and sewn on to a metal flange projecting from the posterior edge of a denture.

(ii) *Kingsley's.*

Double flap of soft rubber exactly fitting and embracing the sides of the cleft.

b. Obturator—

A hard, rigid plug of vulcanite or metal that fills in the cleft and is fixed to the back of a denture.

Forms—

(i) *Suerson's.*

Wedge-shaped plug of vulcanite with base upwards.

(ii) *Kingsley's.*

Large hollow bulb of vulcanite.

A velum is the better appliance with which to learn to speak correctly, but when once this is accomplished, an obturator may be substituted with advantage, as the velum wants frequently renewing on account of deterioration of the soft rubber.

When the appliance is first inserted, the patient must actually be *taught* to speak, which before was impossible. The *immediate* effect of the plate is very little as a rule.

2. ACQUIRED

Causes :—

a. Accidents.
Stabs.
Gun shots, &c.

b. Syphilis.

c. Removal of growths.

Varieties :—

Usually asymmetrical.
Usually perforation anywhere in palate.
Rarely cleft through posterior edge.

Effects :—

Deglutition. } Both interfered with great deal.
Speech. }

Treatment :—

A. Operative—

Plastic operation if feasible and *not* due to syphilis.

B. Mechanical—

a. Simple denture if hard palate only involved.

b. Denture with sheet rubber extension if soft palate involved.

CHAPTER XXII

DANGERS ATTENDING ADMINISTRATION OF NITROUS OXIDE

A. Connected directly with the N_2O

1. **Cyanosis** :—
   Obstructed respiration from—
   - *a.* Tongue falling back.
   - *b.* Tongue swelling.
   - *c.* Growths in mouth or neck.
   - *d.* Œdema of mouth or throat.
   - *e.* Malposition of head.
   - *f.* Tight clothing.
   - *g.* Mucus in air passages, from catarrh.
   - *h.* Vomit in air passages.
   - *i.* Lung disease.
   - *j.* Overdose.
2. **Syncope** :—
   - *a.* Heart disease.
   - *b.* Weak heart.
   - *c.* Alcoholics (bad arteries).
   - *d.* Fright.
3. **Epilepsy.**
4. **Shock** :—
   - *a.* Action of N_2O.
   - *b.* Operating before Vagus centre inhibited.
     N.B.—Vaso-motor centre paralysed before Vagus centre.

5. **Struggling of Patient:—**
   a. Alcoholics.
   b. Neurasthenics.
6. **Apoplexy:—**
   a. Granular Kidney.
   b. Syphilis, &c.
7. **Miscarriage.**

B. Connected with Operation

1. **Foreign Body in Air-passages or Œsophagus:—**
   E.g. *a.* Tooth.
   b. Piece of forceps.
   c. Piece of gag.
   d. Denture.
   e. Blood clot.
2. **Tooth Broken or Forced Out with Gag.**

Precautions against above:—

1. Have ready:—
   a. Tongue forceps.
   b. Mason gag.
   c. Spoon or cloth to catch roots.
   d. Tracheotomy instruments.
   e. Amyl nitrite capsules.
   f. Injection syringe of alcohol or ether.
2. Remove each tooth from mouth as extracted.
3. Do not put gag on loose or frail tooth.
4. Remove dentures.
5. Loosen clothing.
6. Avoid bad patients—
   Lung disease.
   Heart disease.
   Alcoholics.
   Bright's disease, &c.
   Or give them nitrous oxide and oxygen.
7. Avoid meal before operation.

CHAPTER XXIII

HÆMORRHAGE AFTER TOOTH EXTRACTION

Causes:—

1. Laceration with tearing of large vessel.
2. Vessels not contracting—
   - Old age.
   - Syphilis.
   - Inflammation.
   - Menstruation.
3. Blood not clotting—
   - Sea scurvy.
   - Purpura.
   - Hæmophilia.

Varieties:—

1. Primary.
2. Recurrent.

1. Primary

Ordinary hæmorrhage should cease in a few minutes, but if not, means must be taken to stop it, starting with the milder remedies:—

a. Ice-cold water or ice to suck.

b. Styptics on wool in socket for 15 minutes or more.

E.g. Adrenalin chloride.
Tincture of Hamamelis.
Tannic acid.
Gallic acid.

c. Pressure with any of above for few minutes.
If above fail, then proceed as given below for recurrent hæmorrhage.

2. Recurrent

This usually comes on some few hours after the extraction.
First observe whether blood clotting or not.

If *clotting*, give—

a. Ext. Ergotæ Liq. m. xx. every 3 hours; or

b. Injectio Ergotinæ Hypodermica, m. x.

If *not clotting*, give—

a. Liq. Ferri Perchlor. m. xx. every 3 hours; or

b. Acidi Gallici, gr. xv. every 3 hours.

Next observe whether bleeding from—

a. Gums.

b. Socket.

a. **Gums.**

If *capillary* oozing—

Pressure with pad of styptic (see above).

If *arterial* bleeding—

Twist vessel with artery forceps.

If above fail—

Apply pad of styptic and 4-tailed bandage till next morning.

b. **Socket.**

Wash well out and plug tightly at once with one of following:

(i) Matico leaf rolled up.

(ii) Lint, tape, or wool soaked in styptic.

(iii) The extracted tooth.

(iv) Plug of wood, wrapped in lint and styptic.

Keep in place with one of following:—

(i) Pad of lint or cork and 4-tailed bandage.

(ii) Wedge of wood between adjoining teeth.

(iii) Figure-of-eight ligature around adjoining teeth.

Leave for 24 hours.
If still bleeding on removal—replug.
This may be done 3 or 4 times if necessary.
More drastic methods if above fail:—

(i) Actual cautery.
(ii) Trephine inferior dental canal (if for lower tooth), and plug with ivory peg.
(iii) Digital pressure on common carotid artery.
(iv) Tie common carotid artery.

General Directions:—

Whether bleeding from gums or socket—
Avoid all stimulants.
Avoid hot drinks.
Avoid all excitement.
During day, remain sitting with body erect.
During night, recline with head raised.

Hæmophilia

If extractions necessary, give patient—

a. Ext. Ergotæ Liq. m. xx. t.d.s. or
b. Calc. chlor. gr. x. t.d.s.
} For 10 days before.

Operate in morning to give all day for treatment if necessary.

CHAPTER XXIV

DRUGS COMMONLY USED IN DENTAL SURGERY

Actions of Drugs

1. **Anæsthetics** (local) and **Anodynes**—

   Act upon and depress nerve endings, thereby rendering conduction of impulses impossible and therefore preventing or relieving pain.

   Used as injections and local applications.

   Examples—

   Aconite, tincture of.
   Cloves, oil of.
   Cocaine, hydrochloride of.
   Eucaine.
   Ethyl, chloride of.
   Novocaine.

2. **Antiacids**—

   Have direct counteraction to acid conditions in the mouth.

   Used in mouth-washes and tooth-powders.

   Examples—

   Magnesium, heavy carbonate of.
   Sodium, bicarbonate of.

3. **Antiseptics**—

   Prevent putrefaction by arresting growth of, or destroying, micro-organisms.

   Used in mouth-washes, lotions, and applications.

Examples—

Carbolic acid.
Cloves, oil of.
Copper, sulphate of.
Formalin.
Hydrogen, peroxide of.
Iodine, tincture of.
Lysol.
Mercury, perchloride of.
Silver, nitrate of.
Zinc, chloride of.

4. **Astringents**—

a. Coagulate protoplasm of upper layers of cells.

b. Constrict vessels and lessen exudation.

Used in mouth-washes tooth-powders, and applications.

Examples—

Alum.
Copper, sulphate of.
Iodine, tincture of.
Myrrh, tincture of.
Potassium, chlorate of.
Rhatany, tincture of.
Tannic acid.
Zinc, chloride of.

5. **Caustics** (escharotics)—

Destroy the surface of living tissues. When acting upon ends of dentinal fibrils they are rendered insensitive.

Examples—

Arsenious acid.
Carbolic acid.
Copper, sulphate of.
Silver, nitrate of.
Zinc, chloride of.

6. **Counter-irritants**—

a. Dilate superficial vessels, thereby causing free flow of blood through the part, and hence blood and plasma drawn more rapidly through the deeper and inflamed area.

b. Irritate the superficial nerves, and thereby reflexly relieve the pain from the deeper nerves irritated by the inflammation.

Examples—

Iodine, strong solution of (Liq. Iodidi Fortis).

Iodine, tincture of.

7. **Hæmostatics**—

Given *internally*, constrict arteries and lessen frequency of heart, thereby tending to arrest hæmorrhage.

Given by mouth or by injection.

Examples—

Gallic acid.

Ergot, liquid extract of.

Ergotin.

8. **Narcotics**—

Given *internally*, act upon central nervous system, dulling the conduction and reception of impulses, thereby relieving pain.

Examples—

Antifebrin.

Antipyrin.

Phenacetin.

9. **Styptics** (local hæmostatics)—

a. Hasten coagulation of blood by precipitation of albumen.

b. Constrict broken vessel.

c. Coagulate perivascular tissue.

Thereby tending to arrest hæmorrhage.

Used as local applications.

Examples—

Adrenalin, chloride of.
Gallic acid.
Hamamelis, tincture of.
Tannic acid.

Uses of Drugs

Uses.	*Strength or Dose.*	*Action.*
Aconite, Tincture of.		
Periodontitis	Pure (B.P.)	Anodyne
Pulpitis	,,	,,
Adrenalin Chloride.		
Hæmorrhage	1 in 1000	Styptic
Pulp extirpation	1 in 1000 (with cocaine)	,,
Tooth extraction	,,	,,
Alum.		
Inflammation of mouth	Mouth-washes, gr. x. to ʒi.	Astringent
Laceration of mouth	Applications, Glyc. Aluminis (1 in 7)	,,
Ulceration of mouth		
Antefebrin.		
Periodontitis	gr. x. t.d.s.	Narcotic
Pulpitis	,,	,,
Pain from erupting 3rd molar	,,	,,
Neuralgia	,,	,,

	Uses.	*Strength or Dose.*	*Action.*
Antipyrin.			
	As for Antifebrin.		
Arsenious Acid (As_2O_3).			
	Devitalising pulp	gr. $\frac{1}{16}$	Caustic
	Sensitive dentine (far from pulp)	gr. $\frac{1}{32}$	,,
Carbolic Acid (C_6H_5OH).			
	All septic conditions in mouth	1 in 200 to 1 in 60 in mouth-wash	Antiseptic
	After extractions		
	Syringing abscess	1 in 40	,,
	Sterilising instruments	1 in 20	,,
	Tooth-powders	gr. v. to ʒi.	,,
	Mopping out cysts	Ac. Carbolici Liq. (B.P.) = 90%	Caustic
	Painting ulcers		
	Pain after extraction		
	Obtunding dentine		
	Killing pulps of temporary teeth		
Cloves, Oil of (*Oleum Caryophylli*).			
	Pulpitis	Pure	Anodyne
	Exposed pulp	,,	,,
	Sensitive dentine	,,	,,
	Root dressing	,,	Slight antiseptic
	Tooth-powders	m. v. to ʒi.	Flavouring agent
	Mouth-washes	,,	,,

Uses.	*Strength or Dose.*	*Action.*
Cocaine Hydrochloride.		
Extractions . . .	gr. ½ in m. x. injected	Local anæsthetic
Removal of growth .		
Lancing gum . . .		
Alveolotomy . . .		
Opening antrum . .		
„ cyst . . .		
„ abscess . .	Paint, 20 %	„
Root trimming . .	„	„
Impression taking . .	Paint, 5 %	„
Removal of pulp . .	Saturated solution	„
Copper Sulphate ($CuSO_4$).		
Syringing abscess, &c. .	gr. ij. to ℥i.	Antiseptic & astringent
Pyorrhœa . . .	Powdered crystals	„
Follicular stomatitis .	Touch with crystal	Caustic
Root dressing. . .	Powdered crystals	Antiseptic
Ethyl Chloride.		
Extractions . . .	Pure (sprayed on)	Local anæsthetic
Small operations on gums	„	„
Opening abscess . .	„	„
Sensitive dentine (*small cavities*)	Pure (on cotton wool)	„
Eucaine.		
As for Cocaine.		

Uses.	*Strength or Dose.*	*Action.*

Formalin (40 *per cent. solution of Formic Aldehyde in water*).

All septic conditions of mouth	1 in 2000	Antiseptic
Syringing abscess, &c. .	1 in 1000	,,
Sterilising instruments .	1 in 500	,,
Root dressing. . .	40% solution	,,

Gallic Acid ($H_3C_7H_3O_5$).

Hæmorrhage . . .	Pure as application	Styptic
	gr. xv. t.d.s. internally	Hæmostatic

Hamamelis, Tincture of.

Hæmorrhage . . .	Pure as application	Styptic

Hydrogen Peroxide.

All septic conditions of mouth	10 vols. (=about 3 %)	Antiseptic
Syringing abscess, &c. .	10 vols.	,,
Pyorrhœa . . .	20 vols.	,,
Root dressing. . .	20 vols.	,,

N.B.—"20 vols." indicates that 1 volume of the liquid is capable of giving off 20 volumes of oxygen.

Iodine.

Periodontitis . . .	Liq. Iodidi Fort. (1 in 9)	Counter-irritant
Pulpitis	,,	,,
Mopping out sinus. .	Tr. Iodidi (1 in 40)	Antiseptic & astringent
Syringing abscess, &c. .	Iodine water (straw colour)	,,

Uses.	*Strength or Dose.*	*Action.*
Lysol.		
Sterilising instruments .	1 in 50	Antiseptic & lubricant
Magnesium Carbonate (Heavy).		
Oral acidity . . .	gr. xx. to ℥i. (mouth-wash)	Antiacid
Tooth powders . .	℥ij. to ℥i.	Antiacid & cleansing
Mercuric Chloride (*Hydrargyri Perchloridum*).		
All septic conditions of mouth	1 in 5000	Antiseptic
Syringing abscess, &c. .	1 in 1000	,,
Root dressing. . .	1 in 50 (alcohol)	,,
Syringing pyorrhœa .	1 in 500	,,
Myrrh, Tincture of.		
Inflammation of mouth .	m. xv. to ℥i. (mouth-wash)	Astringent
Laceration . . .	,,	,,
Novocaine.		
As for Cocaine.		
Phenacetin.		
As for Antefebrin.		
Potassium Chlorate ($KClO_3$).		
Inflammation of mouth .	gr. x. to ℥i. (mouth-wash)	Astringent
Laceration . . .	,,	,,
Ulceration . . .	,,	,,
Tooth powders . .	℥i. to ℥i.	,,
Stomatitis . . .	gr. i. to x. internally	Specific

Uses.	*Strength or Dose.*	*Action.*
Rhatany, Tincture of (*Tinctura Krameriæ*).		
Inflammation of mouth .	m. ij. to ℥i. (mouth-wash)	Astringent
Laceration . . .	,,	,,
Ulceration . . .	,,	,,
Silver Nitrate ($AgNO_3$).		
Sensitive dentine . .	Pure	Caustic
Erosions . . .	,,	,,
Carious dentine . .	,,	Antiseptic
Exposed dentine . .	,,	,,
Roots before crowning .	,,	,,
Ulceration . .	,,	Caustic
All septic conditions of mouth	gr. ij. to ℥i. (mouth-wash)	Antiseptic
Syringing abscess, &c. .	gr. iv. to ℥i.	,,
Tannic Acid ($C_{14}H_{10}O_9$).		
Hæmorrhage . . .	Pure	Styptic
Application to pulp after As_2O_3	,,	,,
Inflammation of mouth .	*a.* Mouth-wash, gr. iv. to ℥i.	Astringent
Laceration . . .	*b.* Application, Glyc. Ac. Tannici (1 in 5)	,,
Ulceration . . .		
Pyorrhœa . . .	Saturated sol. in spirit	,,
Tooth powders . .	gr. xx. to ℥i.	,,
Zinc Chloride ($ZnCl_2$).		
All septic conditions of mouth	gr. ij. to ℥i. (mouth-wash)	Antiseptic & astringent
Syringing abscess, &c. .	gr. iv. to ℥i.	,,
Sensitive dentine . .	Pure	Caustic
Erosion	,,	,,

Prescription Writing

Superscription.

℞ = recipe = take of.

Inscription.

Names of the drugs, written in Latin in the genitive case, and their quantities. The last drug given is followed by "*ad*," meaning "*up to*," so that a definite quantity is always made up.

Subscription.

Directions to the dispenser, written in Latin.

E.g. Misce.
Fiat collutorium.
Fiat dentifricium.
Fiat lotio.
Fiat mistura, &c.

Signature.

Directions to the patient, generally written in English—preceded by the word *Signa*.

Names, &c.

Patient's and prescriber's names and date.

Abbreviations.

m.—misce.
s.—signa.
āā.—ana = of each.
ss.—semi = half.
ft.—fiat = let it be made.
q.s.—quantum sufficit = a sufficiency.

Example.

Superscription.

℞.

Inscription.

Acidi carbolici. } āā. ℥i.
Tr. Arnicæ. }
Potassii Chloratis, ℥ij.
Glycerini, ℥ss.
Aquæ Rosæ, ad. ℥viij.

Subscription.

Fiat collutorium.

Signature.

s. "Use with an equal quantity of warm water to rinse the mouth frequently."

Patient's name. Prescriber's name.

Date.

CHAPTER XXV

TREATMENT OF HYPERÆSTHESIA OF DENTINE

Manipulation.

Sharp and warm instruments.
Purposive movements—no scraping.
Use much tact, sympathy, and forbearance.

Drugs.

1. **For Immediate Effect :—**

a. Dehydration of Fibrils—

Rubber dam.
Blasts of hot air.
Alcohol. } Applied on wool.
Glycerine. }

b. Cauterisation of Fibrils—

Carbolic (liquid). }
Menthol. }
Carbolic and menthol. } Applied on wool and dried out with hot air.
Zinc chloride (deliquesced). }
Silver nitrate (20% sol.). }

c. Production of Cold—

Chloroform. }
Ether. } Applied on wool.
Ethyl chloride. }

d. Direct Anæsthetic Action—

Oil of cloves.
Oil of cinnamon.
Oil of peppermint.
Cocaine hydrochloride. } Applied on wool.
Novocaine.
Eucaine.
Stovaine.

Cataphoresis and cocaine.
Injection of cocaine into dentine by special syringe.
Injection into gum of—
Cocaine.
Novocaine.
Stovaine.
Eucaine.

2. For Later Effect:—

Carbolic acid—sealed in for few days.
Oil of cloves—sealed in for few days.
Arsenious acid—sealed in for 24 hours (very shallow cavities).
Paraform paste—sealed in for few days. Made as follows—
Paraform—1 part.
Zinc oxide—10 parts.
Carbolic and menthol, q.s. to make a paste.
Oxychloride cement, for few weeks.
Oyxphosphate cement, for few weeks.

General Treatment.

Improve general health.
(Teeth more sensitive when patient unwell.)
Milk of magnesia, if exposed cavities sensitive.

DEVITALISATION OF THE PULP WITH ARSENIOUS ACID

Action of As_2O_3 on the Pulp.

Dilates vessels.

Forms compound with hæmaglobin—arsenhæmaglobin.

Increase in size of connective tissue cells—3 or 4 times their normal size.

Increase of nuclei of neurilemma of nerve fibres, and wasting of axis cylinders.

Pulp and dentine stained brown colour.

Contra-indications to its Use :—

1. When Pressure Anæsthesia can be conveniently used.
2. Open apex. (See Chap. ii. for dates of formation.)
3. When *great* pain has been caused by previous use.
4. Patients susceptible to action of As_2O_3.
   Necrosis.
   Suppuration.
   As. poisoning.
   } Told by previous use.
5. Where it cannot be sealed in.
6. Previous or existing periodontitis.
7. When patient cannot be seen for long time.
8. Patients taking arsenic medicinally may *possibly* be immune to the action on their pulps. (?)

Action Retarded by :—

1. Faulty application—
   Too little.
   Not over the exposure.
   Pushed off by filling.
   Washed out by leaking, &c.
2. Too thick layer of dentine.

3. Chronic inflammation of pulp, especially—
   Pulp stones.
   Secondary dentine.
   Fibroid degeneration.
   Polypus.
   Suppuration of surface.

THE BLEACHING OF TEETH DISCOLOURED BY DEATH OF PULP

Preliminary Procedure.

Prepare cavity.
Clear canal and render aseptic.
Plug apex.
Apply any of following methods, using rubber dam and platinum broaches:—

1. Chlorinated Lime.

Fill canal and pulp cavity with the chloride of lime moistened with 50 per cent. acetic acid.
Seal with G.P.
Renew in 2 or 3 days until bleached.

2. Sulphur Dioxide.

Dessicated sodium sulphite, 10 parts.
Dessicated boracic acid, 7 parts.
Pack in mixed powder, moisten with water, and seal at once.
SO_2 is liberated.

Pyrozone.

Mop out cavity continually for ¼ hour.
Seal little in for few days.

4. **Sodium Peroxide.**

Put saturated solution on asbestos wool in cavity for 10 minutes.

Wash with warm water.

Seal in pyrozone for few days.

5. **Chlorine Water.**

Prepare fresh solution of chlorine gas in water and mop out cavity continually for $\frac{1}{2}$ hour.

CHAPTER XXVI

THE USE OF X-RAYS IN DENTAL SURGERY

As a Means of Diagnosis in the following :—

1. Unerupted tooth—especially lower 3rd molars.
2. Buried root.
3. Orthodontia. *To indicate—*
   - *a.* Presence of teeth :—
     - (i) Whether extracted.
     - (ii) Whether not erupted.
     - (iii) Whether undeveloped.
   - *b.* Direction of roots.
   - *c.* If temporary persists, whether permanent is near.
   - *d.* Whether root of temporary is absorbed.
   - *e.* Presence of unerupted supernumerary.
4. Exostosis of root.
5. Absorption of root.
6. Pulp stones.
7. Odontomes.
8. Bony growth of jaw.
9. Fracture of jaw.
10. Dislocation of jaw.
11. Presence of abscess cavity, when not apparent clinically.
12. Presence of cyst.
13. Foreign bodies in the antrum.
14. Salivary calculus in ducts or gland.

Methods of Using.

Outside Method—

Plate placed outside cheek of side required.

Tube placed in such a position on the other side of patient that—

a. For the mandible, the rays pass obliquely upwards through floor of mouth, thus avoiding the side of jaw not required.

b. For the maxilla, the rays pass between the teeth of the opposite side, with the mouth propped wide open.

Inside Method—

Protected film placed inside mouth and held against the jaw.

Tube placed outside.

Especially useful for incisor region.

A tube has been made that can be used inside the mouth, with the plate outside, but it is necessarily very small, and not of much practical value at present.

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